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SPONTANEOUS HYPERINSULINISM*

Report of a Case of Hyperinsulinism Cured by Surgical Intervention

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THE clinical syndrome of spontaneous hyperinsulinism has been recognized as a disease entity since 1927. The first case with complete laboratory and clinical studies including operative and necropsy findings was reported by Wilder, Allan, Power and Robertson from the Mayo Clinic[1]. Since that time other cases have been reported from the same and other clinics. A complete review of these cases may be found in the papers by Howland, Campbell, Maltby and Robinson[2] who reported the first instance when a cure was effected by surgical removal of a carcinoma of the islet cells of the pancreas; by Womack, Gnagi and Graham[3] who reported a similar case and by Allan, Boeck and Judd[4] who discuss the surgical treatment of hyperinsulinism. These latter authors state that when the disease is due to a localized tumor of the pancreatic islands a complete cure may be expected by its excision.

Since the present case†† was entirely cured by excision of a portion of the pancreas which included an islet cell adenoma it seems worth while to report the medical, surgical and pathological findings in some detail.

The occurrence of unquestioned adenomas of the islands of the pancreas has been reported a number of times. Warren[5] in 1926 collected 16 such cases from the literature to which number he added four more. Since that time one additional case has been reported by Natali[6]. All of these adenomas were found during necropsy studies and in none of those where a clinical report or the cause of death was mentioned was there evidence to suggest that they might have caused symptoms.

REPORT OF CASE

History: Mrs. A. B., aged 56 years, an intelligent white American saleswoman began in 1924 to notice periods during which she would feel faint and the world would "go black." At the start the attacks came on at some interval after meals, usually in the evening, and were associated with overfatigue. They were always accompanied by profuse perspiration. She noticed early that, if some one spoke to her or she could get a glass of malted milk when the attack threatened, it could be aborted. She did not lose consciousness and was able to continue with her work for about a year when she fell in a subway car and suffered minor injuries to her head and thigh. By now her attacks had become gradually more frequent and more severe and compelled her to stop working. A typical attack at this time began with a numbness of the upper lip radiating to the spine and down the arms. Malted milk taken immediately would abort the attack, otherwise it progressed into dizziness, faintness and inability to walk straight. An attack usually lasted about half an hour during which time she was unable to move or speak though she would not lose consciousness. With one exception she never bit her tongue, frothed at the mouth, uttered cries or suffered a loss of sphincter control. She always felt exhausted for an hour or more after an attack terminated. She also noticed that eating at frequent intervals seemed to decrease the frequency of the attacks.

The patient first attended the out-patient department of this hospital May 14, 1927 with complaints of tiredness and exhaustion. Prior to this she had been cared for by private physicians or in another hospital out-patient clinic. She made seven visits to this hospital between May 14, 1927 and January 10, 1928 at which times in addition to physical examinations which were always essentially negative she had repeated x-ray studies of her skull and a metabolism determination May 18, 1927 which was +28 per cent from the expected normal. Stereoscopic plates of the skull showed no definite evidence of disease. The diagnosis during this time lay between neurasthenia, epilepsy and cerebral tumor. The first-named disease was considered the most likely and the patient was treated accordingly.

At her last visit January 10, 1928, she stated that, in addition to her previous symptoms, she had had repeated attacks of vomiting, chiefly in the morning, for the preceding two weeks. She was advised to enter the hospital but desired to return home and was not seen again for almost two months.

Admission to Hospital: Patient was admitted to the hospital for the first time March 5, 1928 complaining of attacks of unconsciousness. Further notes of interest are the following: Patient is one of a family of 12 children. Her father, mother and one brother died of probable heart disease at the ages of 83, 75, and 70 years respectively. Otherwise her family history for disease is irrelevant.

The patient in early life had practically every illness of an infectious nature including tonsillitis, pneumonia and scarlet fever. The last named, at the age of seven years, left her with chronic bilateral otitis media which has drained intermittently since; hemorrhoids present for the past few years have never given her any trouble. Her menstrual

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††Referred to by Dr. Harvey Cushing in The Lister Lectures, Lancet 2-119 (July 19) 175 (July 26) 1930.

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history was normal with menopause established uneventfully at the age of 51 years. Her appetite had always been fairly good. Her usual weight was 135 pounds until the onset of the present illness, since which time she has gained 50 pounds. She attributed this latter to her unusual consumption of malted milk for which she had spent $4.00 to $6.00 weekly for some time. She was always subject to frequent headaches but did not think they had changed during her present illness or were associated with it in any way. For some years she had had palpitation of the heart upon exertion for which she took small doses of digitalis at intervals. Recently slight dyspnea has accompanied her palpitation.

Physical Examination: The patient was a well-developed and moderately obese woman who was mentally clear and appeared normal for her age. All teeth were replaced by dentures. Her heart was enlarged slightly but otherwise normal; blood pressure was 160 mm. of mercury systolic, and 95 mm. diastolic. Neurological examination including the fundi showed nothing abnormal. There was a marked vasomotor instability as shown by an erythematous blush over her neck and in the skin of the abdomen after stroking. The urine, both single and 24 hour specimens, was of a low specific gravity but was otherwise normal. Stool was normal. Blood serum Wassermann was negative. The red blood cell count was 4,880,000; hemoglobin was 90 per cent; the white cell counts were 13,100 and 11,500 on two occasions with a normal distribution of the cell types. Temperature, pulse and respiration ranges were within normal limits during her stay in the hospital. An electrocardiogram showed left ventricular preponderance but was otherwise normal. Stereos of the skull showed again no evidence of an intracranial tumor. Fasting blood sugar determinations on two different occasions showed 65 and 58 mg. of sugar per cent.

This last finding together with the fact that her attacks were accompanied by fatigue suggested to one member of the staff a similarity to the condition found in athletes after strenuous exertion and which has been shown to be due to hypoglycemia[7]. Unfortunately the patient had no attack during her six days in the hospital despite the fact that she was kept up and active about the ward and on two different days her evening meal was delayed about two hours. A sugar tolerance test was not done. She was discharged from the hospital with the main diagnosis hypoglycemia, cause unknown. She was advised to take an obesity diet and to take a lump of sugar during the morning, afternoon and at any other time when she felt an attack impending.

Interval history: Patient was not seen again until her second admission to the hospital March 24, 1930. However, in response to a letter of inquiry in April, 1929, her reply was "I have not been so well since leaving the hospital. Unconscious spells more frequent—at present I am in the accident ward of the Massachusetts General Hospital as a result of falling and breaking my leg while having a bad spell. No new symptoms."

Readmission to Hospital: Patient stated that for almost a year after leaving this hospital in March, 1928, there had been little change in her condition and that she was able to do most of her housework; that she was able to remain free of severe attacks by rigorously following the practice of taking a large glass of malted milk at 3 p.m., 5 p.m. and 9 p.m. daily in addition to her regular meals; that she had remained weak and often became dizzy and nauseated.

On March 5, 1929, she awakened with her first severe headache for over a year; she ate no breakfast and by noontime felt very faint. On rising and attempting to procure food she fell and broke her left leg just above the ankle. Her next two months were spent in the Massachusetts General Hospital. Excerpts from her case record there show that in view of her history of repeated "spells" she was given luminal grains 1½ twice daily; that she had no spells during the latter part of her stay and that her fracture united uneventfully.

Since that time the patient has had to adhere to her previous régime of three glasses of malted milk daily in addition to her meals. Should the taking of any one of these be delayed for even a few minutes dizziness, sweating, numbness and irritability would appear. A glass of malted milk would bring her back almost to normal instantaneously.

Her own interval story as given above was amplified by her sister who stated that three days before the present admission, when sitting down to the evening meal, the patient complained suddenly of pain in her stomach, weakness, dizziness and nausea, and rubbed her abdomen, this, in spite of the fact that she had had her usual malted milk two hours previously. She walked to the bathroom where she vomited several times within half an hour and became gradually so weak that she slumped down on to the floor. Though still able to answer questions slowly, she had to be carried to her bed. Here she became restless, excited and unruly. She screamed and all attempts to quiet her were resisted until she was given morphia subcutaneously. Following this she slept until 11 p.m. when she arose and had tea and toast in addition to her usual glass of malted milk. She denied recollection of any event from the time she went into the bathroom until awakening at 11 p.m. It was said that the patient had recently developed the habit of poking people in the ribs and other peculiar acts during an attack so that her relatives had begun to fear her. Due to the increasing frequency of the attacks with helplessness and irresponsibility she has spent the past few weeks living with her sister.

Clinical findings at this time were essentially the same as those found on her previous admission. The one noteworthy change was the finding of a fasting blood sugar of 31 mg. per cent (Folin—Wu). Repeated x-ray studies of the skull, chest, abdomen and gastrointestinal tract showed nothing abnormal. The metabolism rate was +2 and when repeated later —4 per cent from normal. In view of our inability to find any demonstrable lesion to explain this patient's symptoms and their similarity, including the associated hypoglycemia, to those of the cases reported by Wilder and associates[1] and Howland and associates[2] we concluded that she suffered from periodic attacks of hyperinsulinism secondary to a tumor of the islet cells of the pancreas. As she preferred to have further observation and attempted treatment before submitting to surgical intervention she was kept in the medical ward. While here, various studies, a few of which will be detailed below, were carried out.

Clinical course in the hospital: Soon after admission lumbar puncture was done, the mechanics of which and the spinal fluid obtained were normal in all respects. During her 32 days in the medical ward eleven fasting blood sugar observations were made using the Folin—Wu method. The values of the eleven determinations ranged from 21 to 50 mg. of sugar per hundred cubic centimeters of blood with one exception when 62 mg. were found. Blood was obtained during a convulsion on two dif-

ferent occasions, the sugar values of which were 25 and 46 mg. per cent.

It is interesting to note that no absolute value for the blood sugar can be given at which level a convulsion would occur. The fasting blood sugar on five different occasions was found to be 31 mg. per cent or lower and yet on each of these occasions the patient seemed quite normal. Soon after admission blood sugar determinations were made at two hour intervals for a period of 24 hours. The values found and their relation to the taking of food are shown in table 1.

TABLE 1

BLOOD SUGAR VALUES AND ASSOCIATED DATA DURING A 24-HOUR PERIOD

Time	Blood sugar in mg. per hundred cubic centimeters	Remarks
5 P.M.		Supper*.
6 P.M.	34	
8 P.M.	53	
10 P.M.	24	Faint, did not respond normally.
12 P.M.	55	
2 A.M.	64	
4 A.M.	40	
6 A.M.	42	
7:15 A.M.		Breakfast.
8 A.M.	33	
10 A.M.	39	
12 noon	42	Dinner.
2 P.M.	74	
4 P.M.	25	No complaints, appeared normal.

*No food given between meals.

During her first few days in the hospital no food was allowed other than her three regular meals. As pointed out above there was no absolute correlation between the level of blood sugar and the patient's general condition. At 10 p.m. in the 24 hour observation period the blood sugar was 24 mg. per cent at which time the patient did not respond normally to questions. No form of therapeusis was used and yet two hours later the blood sugar had risen to 55 mg. and the patient's response was normal. Again at 4 p.m. the following day a value of 25 mg. was found at which time the patient seemed entirely normal.

TABLE 2

SUGAR TOLERANCE TESTS DONE ON DIFFERENT DAYS

Time in hours	92 gm. of glucose by mouth		95 gm. of glucose intravenously	
	Blood sugar mg. for each 100 cc.	Urine sugar	Blood sugar mg. for each 100 cc.	Urine sugar in gm.
Fasting*	26		42	
½	72		211	
1	31	None	87	2.4
2	78		27	0.2
3	71	None	39	

*The sugar was given immediately after withdrawing the fasting blood specimens.

Sugar tolerance tests: The findings in this case differed in one important point from those reported by Wilder and Howland referred to above[1, 2]. These authors found that their patients showed sugar tolerance curves similar to those found in diabetic patients. Sugar tolerance tests were done on different days giving 92 gm. and 95 gm. of glucose by mouth and into the vein respectively. The results of these tests are shown in table 2.

This patient's ability to utilize glucose was what one might expect theoretically. This point was illustrated more emphatically when the glucose was given by intravenous route. As can be seen in table 2 the blood sugar rose from a value of 42 mg. immediately prior to the giving of 95 gm. of glucose intravenously to a high of 211 mg. 30 minutes later. Following this there was a very rapid fall to well below the fasting level at the end of two hours. One can only assume that this patient was able to store glycogen in large amounts. While 2.6 gm. of glucose, a negligible amount, was excreted in the urine during the intravenous test, none appeared when the glucose was given by mouth.

Effect of 100 gm. of glucose intravenously on the respiratory quotient: This experiment was done with the patient in the fasting state. At this time the blood sugar was 46 mg. per cent and the respiratory quotient 0.72. She was then given 100 gm. of glucose intravenously. The respiratory quotient rose to 0.99 and 1.12 after an interval of 40 and 80 minutes respectively. Unfortunately blood taken for sugar determination at the end of 65 minutes was lost. The blood sugar value at the end of two hours was 97 mg. per cent.

Effect of 1 mg. of adrenalin subcutaneously: The fasting blood sugar value was 56 mg. per hundred cubic centimeters before the adrenalin was injected. Blood sugar determinations were made at 15 minute intervals for 60 minutes and again at the end of 90 minutes. There was no appreciable change from the fasting level to be noted. Pulse rate and blood pressure observations, taken coincidentally with each drawing of blood, failed to show the usual physiological response to adrenalin. Hence there may be some question as to the potency of the adrenalin used. The results of this test are reported for purposes of completeness only.

Effect of Atropine: Britton[3] has shown that stimulation of the right vagus nerve in cats prepared properly causes an increased secretion of insulin with an accompanying fall in the blood sugar level. These latter conditions being present in this patient, an attempt was made to correct them by the administration of atropine. A blood sugar determination made several hours after the taking of food was found to be 38 mg. One half a mg. of atropine sulphate was then injected subcutaneously and three further sugar determinations were made at 20 minute intervals. There was a rise to 49 mg. at the end of 20 minutes, then a gradual fall to 42 mg. at the end of an hour. At this time the patient was given 0.5 mg. of atropine sulphate and 25 gm. of glucose intravenously. The following blood sugar values were 117, 74 and 68 mg. at the end of 20, 40 and 60 minutes respectively. This is the sort of curve one would expect had the sugar alone been given intravenously.

The patient was then given 2 cc. of tincture of belladonna by mouth four times daily for a period of five days. During this time no noteworthy change in either the fasting blood sugar or her general condition was observed. She tended toward an attack as readily as previously if food was withheld.

It may be noted that other chemical values of the patient's blood such as the total non-protein nitrogen and its various fractions, calcium, and phosphates were all within normal limits.

During the patient's 32 days under the medical service, her attacks tended to become more fre-

quent and more severe. Although after her first four days in the hospital she was given a glass of malted milk during the forenoon, afternoon, and late in the evening she had to be watched carefully to prevent the occurrence of an attack. Whether this increased tendency to attacks was in any way related to her treatment or was purely a natural progression of her illness it is impossible to state. Since one of these severe attacks, which lasted 20 minutes and necessitated the administration of glucose intravenously to restore her to normal, occurred during the night, the patient grew anxious about her condition and elected to have an exploratory operation in the hope that a localized tumor of the pancreas, which could be removed completely, would be found. After surgical consultation (with Dr. Francis C. Newton) she was transferred to the surgical service with the diagnosis of hyperinsulinism, due probably to an adenoma of an island of Langerhans.

Operation: Exploration of the pancreas was done on May 6, 1930. Under ether anesthesia, a portion of the pancreas, for a distance of 7 cm. from tail to point of transection, containing an adenoma, was removed. With a hypodermoclysis of 10 per cent glucose running continually throughout the operation, a long left rectus incision was made from costal margin to slightly below the umbilicus, carried inward at its upper border toward the ensiform and widened along the border of the ribs of the left side in "T" shaped fashion. This permitted an excellent exposure, particularly of the upper left abdomen. The patient was quite stout and had a very thick panniculus and omentum, and all intra-abdominal structures were unusually fatty. Exploration of the liver failed to show any evidence of metastatic tumor. The gastro-colic omentum was incised directly over the pancreas, coming down on what appeared at first glance to be a normal gland. There was one small area about 6 cm. from the tip of the tail of the pancreas which did not have the same appearance as the surrounding tissue, but we could not feel certain that this might not represent a small hematoma. A resection of the tail of the pancreas to a point just beyond the small suspicious area just described was decided upon. In the process of stripping the pancreas free from surrounding areolar and connective tissue, only one large blood vessel was encountered running along the upper side of the pancreas. This vessel was tied. The smaller vessels were twisted off with the finger tips. In only one place was there any bleeding which caused real difficulty, this occurring just as we transected the pancreas beyond the small tumor mass. The cut end of the pancreas was whipped over and over with running catgut sutures in an effort to close in all leaking points in the gland and avoid the development of a pancreatic fistula. In view of Finney's experience in his first case of pancreatic resection in 1928, in which he was sorry that any drain had been left in the region of the transected gland, the abdomen was closed without drainage.

The operation lasted about two hours, during the major portion of which time there was practically no recordable blood pressure. Just before operation, her blood pressure was 150/80. Within a few moments after the closure had been completed, her systolic reading returned to 90 and her diastolic to 70. We were a little concerned as to the possibility of postoperative hemorrhage inasmuch as, with practically no recordable pressure during the resection, our opportunity to identify possible bleeding points was lessened considerably. Her pulse, however, remained quite steady throughout the operation, and her condition was good at the conclusion. During the operation about 60 grams of glucose were given in approximately 700 cc. of saline by hypodermoclysis. Her blood sugar reading directly after operation was 300 mg. per 100 cc.

Postoperative course: The patient remained in the hospital 31 days after operation. Her convalescence was uneventful in all essentials except for a bronchopneumonia which occurred within three days, chiefly in the right base. Her urine for two days following operation showed the slightest possible trace of sugar, but there was no diacetic acid or acetone. Careful blood sugar determinations were done three times in the first twelve hours, and daily thereafter during her stay in the hospital. See table 3. When patient was well along in her convalescence blood sugar values were determined during a 24 hour period. See table 4. Sugar tolerance test was repeated also giving but 25 gm. of glucose by mouth. See table 5. It is interesting to com-

TABLE 3

POSTOPERATIVE BLOOD SUGAR DETERMINATIONS

Date	Time	Blood Sugar
5-6-30	3:00 P.M.	176 mg.
5-6-30	7:00 P.M.	253 mg.
5-7-30	8:00 A.M.	156 mg.
5-8-30	8:00 A.M.	142 mg.
5-9-30	8:00 A.M.	200 mg.
5-10-30	8:15 A.M.	155 mg.
5-11-30	8:30 A.M.	152 mg.
5-12-30	8:00 A.M.	132 mg.
5-13-30	8:00 A.M.	136 mg.
5-14-30	8:00 A.M.	118 mg.
5-15-30	8:00 A.M.	148 mg.
5-16-30	8:00 A.M.	130 mg.
5-17-30	8:30 A.M. (30 min. p. c.)	140 mg.
5-18-30	8:00 A.M.	148 mg.
5-19-30	8:00 A.M.	151.5 mg.
5-20-30	7:30 A.M. (fasting)	129 mg.
5-21-30	8:00 A.M.	93.8 mg.

TABLE 4

BLOOD SUGAR DETERMINATIONS DURING A 24-HOUR PERIOD

Date	Time	Blood Sugar
5-23-30	12:40 A.M.	139 mg.
" " "	3:00 A.M.	122 mg.
" " "	6:30 A.M.	119 mg.
" " "	Breakfast 8:00 A.M.	
" " "	9:00 A.M.	169 mg.
" " "	12:00 noon	134 mg.
" " "	3:00 P.M.	126 mg.
" " "	Supper 5:00 P.M.	
" " "	7:00 P.M.	165 mg.
" " "	10:00 P.M.	129 mg.

TABLE 5

SUGAR TOLERANCE TEST

Date	Time	Blood Sugar*
5-25-30	8:00 A.M.	115 mg.
" " "	8:20 A.M.	(given 25 grams of glucose)
" " "	8:50 A.M.	150 mg.
" " "	9:20 A.M.	176 mg.
" " "	10:20 A.M.	132 mg.
" " "	11:20 A.M.	132 mg.

*Urine specimens after each blood sugar, all negative.

pare these results with those found previous to operation.

On the twenty-seventh of May, 21 days after operation, with the patient up and about, a metabolism test was done which showed a +1 reading.

On discharge from the hospital, the patient felt perfectly well physically, and had been up and about for a week. Her blood sugar had subsided to normal on a regular house diet. The attacks which had been a daily occurrence previous to operation had been totally absent since operation.

PATHOLOGICAL REPORT

Gross Examination: Specimen, which weighed 16½ gm., consisted of the terminal 7 cm. of the pancreas. On the anterosuperior border 1 cm. from the resected margin was an oval, slightly elevated, reddish grey mass somewhat firmer in consistency than the surrounding pancreatic tissue. A cross section passing through this mass showed it to be ovoid shaped and to measure 1.6 x 1.2 cm. (Fig. 1). Its cut surface was reddish brown in color and finely lobulated. The tumor was completely encapsulated and easily separable from the adjacent tissue by blunt dissection. The pancreatic tissue itself was of normal appearance.

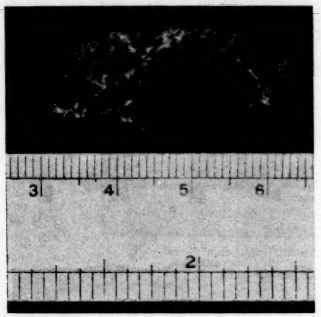

FIG. 1. Cross section of pancreas passing through the adenoma, of island of Langerhans type.

Physiological Examination: A fragment of fresh tumor tissue weighing 180 mg. was macerated in 3 cc. of normal salt solution, centrifugated and 1 cc. of supernatant fluid injected intravenously in a rabbit. Blood sugar determinations were made before and at half hourly intervals after the injection. These values were 170, 70, 90 and 120 mg. per cent respectively. Thus, it can be seen that the tumor was very rich in insulin or a substance which gave a pronounced insulin-like effect.

Microscopic Examination: The tumor was composed of epithelial cells supported by a vascular stroma with but a very slight amount of connective tissue (Fig. 2). This arrangement presented the general architecture of glands of internal secretion. The capsule of varying thickness was composed of loose fibrous tissue with occasional areas of herniation. There was slight compression of the adjacent pancreatic tissue. The tumor cells on the whole were somewhat larger than those of the islands of Langerhans and among them one occasionally encountered cells in various stages of degeneration. Some of these degenerated cells showed small spaces filled with débris, while others had hyaline droplets and vacuoles in their cytoplasm (Fig. 3). No mitotic figures were found although cells with hyperchromatic nuclei were not infrequent. Special fixation and staining revealed, in the better preserved areas, cells with varying numbers of basic staining granules; in places the cytoplasm was packed with them. Acid granules, however, were not encountered. The islands of Langerhans both adjacent to and distant from the tumor, had a few cells with acid staining granules but none of the basic staining variety. Granular cells, however, were comparatively rare. Islands were present in normal numbers and showed no histological evidence of degeneration or atrophy. The acinar and duct elements revealed no morbid changes.

COMMENT

This is the oldest patient (57 yrs.) on record in whom a neoplasm arising from an island of Langerhans has been found. It is one of the few cases where the benignity of the tumor is unquestioned. The only other instance of a simple adenoma was reported by Carr, Parker, Grove, Fisher and Larimore[9] and occurred in a

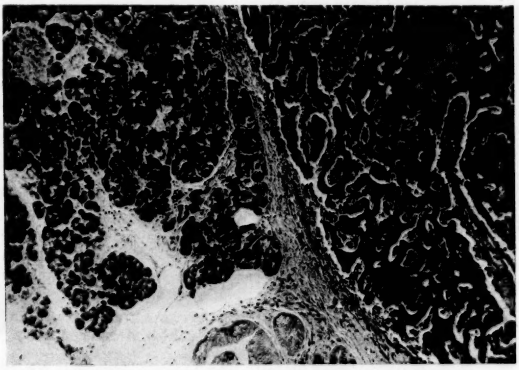

FIG. 2. A low power view to give the general architecture of the tumor, its relation to the pancreas and an island of Langerhans. Eosin-Methylene Blue.

boy of 19 years. As stated by them one might have anticipated finding a malignant tumor in our case due to her comparatively advanced age.

The diagnosis in this as in other cases was difficult. She was looked upon as a case of neurasthenia until her second admission to the hospital (March, 1930) when the symptoms were more pronounced and other similar cases had been reported. At this time the syndrome, including the low blood sugar content, was so complete without any other plausible explanation that a diagnosis of hyperinsulinism was unavoidable.

There is excellent evidence that this patient's troubles were caused by the tumor. First, because her symptoms were identical with those due to an overdose of insulin; this tumor was exceedingly rich in that substance. Secondly, because she has been entirely cured by its removal. On January 22, 1932, over 20 months after operation her fasting blood sugar was 70 mg. per cent and she was physically well and able to care for her own home. She is quite comfortable on three small meals daily.

The resection of the distal portion of the pancreas presents no unusual risk or problem.

The present case demonstrates very well that there is no critical level of blood sugar at which a patient will develop symptoms.

FIG. 3. A portion of the tumor showing relation of cells to blood vessels. Note the scattered cells with vacuolization, some adjacent to blood vessels. Eosin-Methylene Blue.

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VITAMINS REQUIRE AID

Reports from Montreal indicate that Dr. William B. Castle of Boston has found that vitamins are apparently helpless in improving health conditions without the aid of a compensatory agent. This, he has found, exists in the stomach and appears to prepare the way for an understanding of how food may affect disease. Dr. Castle's report was submitted before the American College of Physicians at a recent meeting. The experiments which have led to these conclusions have been conducted in the Boston City Hospital, especially in dealing with such deficiency diseases as anemia, rickets, scurvy and beri-beri. This description of discoveries by Dr. Castle was given in the John Phillips Memorial Prize Oration and is one of the important contributions to medicine of which Boston may well be proud.

THE DEATH OF SIR FREDERIC HALLETT

The *New York Times* reports that Sir Frederic Hallett, for thirty years Secretary of the Imperial Cancer Research Fund, died February 6, 1933, at the age of seventy-two. He resigned last November because of failing health.

Sir Frederic was knighted in 1928 for services in the British Army and Navy during the World War.

His important contributions have been in cancer research.

THE ROLE OF INFECTION IN BURNS*

The Theory and Treatment With Special Reference to Gentian Violet

BY ROBERT HENRY ALDRICH, M.D.†

The author wishes it to be understood that to Dr. W. M. Firor, Associate Professor of Surgery in the Johns Hopkins Hospital, belongs the full credit for conceiving the infectious theory in the symptomatology of burns and the treatment of the latter with gentian violet as herein described. Due to his absence from this country, he was unable to follow up this problem fully, and delegated the privilege of publication to the author. The first cases studied were with Firor at the Johns Hopkins Hospital and the later ones were patients on the Harvard Surgical Teaching Service of the Boston City Hospital.

THIS paper deals chiefly with modern conceptions regarding the physiology, chemistry, pathology, and treatment of burns. There is no attempt made to present a full history of any of these aspects, as a brief review of the literature reveals a certain cyclic recurrence of ideas and treatments, and also, during the last few years, a sufficiently conclusive group of theories, experiments and panaceas.

Each new trend in medical progress has had some effect on the general ideas concerning burns. These new ideas were plausible at the time, but when subjected to careful analysis and experimentation, fell into disuse or were clung to out of habit or lack of a better substitute. Each of these theories carried with it a form of treatment which answered the pathology of the condition as it was understood at the time, but the majority of these theories are now so obsolete as to have no value save in an historical sense, and therefore will not be discussed here. There are, however, two theories which must be considered and evaluated, and a third which will be mentioned briefly before it is discarded.

Any theory, to be of value, must be able to explain the various phenomena encountered in a burned patient, from the termination of the primary shock (which, in the opinion of the author, is an entity in itself similar to any other surgical shock), through the patient's hospital course, which is characterized by fever, leucocytosis, blood changes, secondary infection, and the other well-known sequelae, and the gross and microscopic specimens from postmortem examination.

A theory which will be considered briefly supposes that the destruction of the functions of the skin is the causative agent of the symptoms and pathology of burns; the functions included here being that of excretion, heat regulation, and sensation. A variety of ingenious experiments have been carried out along these lines. The factor of heat regulation has been studied by keeping the burned animals at different temperatures; this however has always proved unsatisfactory, for it has been shown that in spite of being supplied with the optimum temperature the animals still exhibited the usual symptom complex associated with burns. Likewise rabbits have been burned over the entire extent of both ears, with and without section of the sensory nerves, with no variation between the two groups[16]. There have been no marked changes in the catabolic substances in the blood when the skin has been experimentally robbed of its excretory function, such as in the gilding of the entire body[11]. In the face of these results it is impossible to consider the destruction of skin of vital importance.

The two most widely accepted theories are:

1. The theory which supposes that there is some toxin liberated from the burned area which makes its way into the blood stream and produces a toxemia.

2. The theory which explains the phenomena on the various known changes in the blood stream and in the altered function and composition of the blood.

The theory of toxemia has been advocated by a large group of investigators. It is based on the assumption that there is formed in the site of the burn, or in the burn itself, a toxic substance which is responsible for the general reaction of the patient. Many substances described by observers have been found in the blood, in the burned skin, and in the urine. The variety of substances is so great, including as it does pyridine, guanidine, peptone, primary and secondary proteoses, histamine and ptomaines, that it indicates a lack of agreement as to the actual toxin; added to this is the fact that few of the investigators have been able to check the results of others.

Reiss[20], Lustgarten[15] and Spietschka[25], all succeeded in finding various substances in the urine of burned patients which were toxic when injected into animals. There was, however, no unified opinion as to what these were, nor were they always present.

Greenwald[10] reported two fatal burns in chil-

*From the Harvard Surgical Teaching Service of the Boston City Hospital.

Read at a meeting presented by the Harvard Surgical Teaching Service of the Boston City Hospital in February, 1932.

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dren, in which death occurred with hypoglycemia, there being no description of the burns themselves. He then carried out some experiments on rabbits in which he found that all of his animals died with hyperglycemia. He described the cause of death as being due to two factors, the first pure shock ensuing soon after the burn, the second, coming on later, supposedly caused by a degeneration of the adrenals. Again he did not describe the burns at all, except to give their depth and extent.

Olbrycht[17] confirms the reports emphasizing the degenerative changes in the adrenals, his animals also dying in hyperglycemia. He believed these changes due to the burned tissues, and advised débridement of the burn and injection of adrenalin. Again there is no description of the burns.

Bardeen[1], in a study of five fatal burns in children, dying from four to nine hours after being burned, observed degenerative changes in the liver, spleen, kidney, and bone-marrow, with general edema of all the lymphoid tissue, the edema being most marked in the germinal centers. He describes this picture of the lymph glands as being practically identical with that seen in patients dying with an acute infection. He concludes that he has found enough pathology to eliminate any theory other than the one of toxemia. His paper, however, was published long before Underhill[29] had shown that burns over the abdomen and chest cause an elevation of intra-abdominal temperature to as high as 110 degrees and one wonders whether this factor might not have played an important rôle in the tremendous shock and sudden death he observed in his patients.

Pfeiffer[18] was able to isolate split proteins from the burned skin, and found these to be neurotoxic and necrotoxic. He worked out their chemistry, and described them as being soluble in water, alcohol and glycerole, and insoluble in chloroform and ether.

Robertson and Boyd[21, 22] claim that there is found in the skin of burned animals a toxin which "circulates in the blood, either in or adsorbed by the red blood corpuscles, and which causes the symptoms seen in bad superficial burns, and in some cases, death". . . . "The toxic substance consists of two portions, one of which is thermostabile, diffusable, and neurotoxic; the other is thermolabile, colloidal, and necrotoxic. Chemically, the toxin consists of a primary and secondary proteoses." They also state that an extract of normal skin prepared as a control is not toxic when injected into a normal animal, and further, that whole blood from a burned animal when injected into a guinea pig will cause a toxic syndrome which corresponds exactly to that seen in a burn. They admit that boiling of the extract tends to weaken its potency as a neurotoxic agent, in spite of the fact that they specifically state that the toxin is thermostabile.

Underhill and Kapsinow were unable to conceive of a toxin derived from burned areas, and repeated the experiments of Robertson and Boyd, duplicating them in every detail. They confirmed the results above described, in that an extract of burned skin prepared according to the technique of Robertson and Boyd was toxic when injected into guinea pigs. They also obtained the same results by preparing and injecting normal skin in the same manner. Still unable to believe in a skin toxin, they analyzed the extract, which is alcoholic, and found a sufficient quantity of alcohol to account for the symptoms of the animals. As a control, this same quantity of alcohol was injected into the guinea pigs with exactly the same toxic results. This observation leads one to believe that the primary and secondary proteoses discovered by Robertson and Boyd was simple ethyl alcohol. They also cast doubt on the toxic content of whole blood from a burned patient by repeating the experiments, finding no toxicity resulting.

In his report on the tannic acid treatment of burns, Davidson[9] concluded that he was dealing with some toxin, and thought that by coagulating the entire burn, he was preventing absorption. He states, in his paper, that in those cases in which he obtained a good eschar there were very few untoward symptoms even in severe burns. This finding held true so long as the eschar remained dry, but as soon as the eschar showed signs of softening, or where there was underlying infection, there was an increase in temperature and the usual symptoms and physical signs of toxemia were exhibited. It will be seen later that this statement is less an argument for the toxic theory than against it.

It is thus seen that all the evidence in support of the theory of the absorbed toxins is indefinite or contradictory. There seems to have been no agreement as to the nature of the toxin, or the manner in which it works. Many of the experiments are refuted by repetition. The evidence which remains is only clinical observation and can in no way be considered proved. The newest and most convincing experiment to undermine the theory of toxemia is that of Underhill, Kapsinow, and Fisk[29]. They injected trypan and methylene blue into animals after they were burned, and found that there was an increased capillary permeability from the blood into the edema fluid of the burn, also a diminished rate of absorption of the dyes from the edema area back to the blood. In fact, they found that substances which rapidly pass from the blood into the injured area, were not reabsorbed to a significant extent, if at all. To complete this experiment, they injected lethal doses of strychnine both into the burn and the periphery of the burned area, and found that absolutely no symptoms of poisoning were ex-

hibited by the animals. It seems conclusive, therefore, that if such substances as trypan blue and strychnine were not absorbed from a burned area, no vague toxin would be picked up by the blood stream in sufficient concentration to cause the symptoms and signs accounting for the so-called toxemia of burns.

The second theory to be considered, is that one based on the changes in the blood following burns. This problem has been very completely and brilliantly worked out by Underhill, Carrington, Kapsinow and Pack[27]. They found that in extensive superficial burns there was a concentration of blood, and that this concentration has a great deal to do with the outcome of the patient. Their work in this experiment was carried out on twenty-one victims of a local theatre fire. As long as these patients stayed in the hospital, their blood pictures were followed. A summary of the findings showed a marked increase in hemoglobin and a distinct decrease in the blood chlorides. With the concentration of the blood, there was a fall in blood pressure and a lowered temperature. It was also observed that by the restoration of normal blood concentration, there were marked evidences of improvement, and that the development of the usual sequelae of extensive burns was checked. Somewhat later, Underhill, Kapsinow and Fisk[29] carried on further work as to the mechanism of blood concentration. It was shown that a superficial burn involving approximately one-sixth the total surface area of the rabbit, induced within a period of six hours a significant loss of fluid from the blood in producing a subcutaneous edema. The fluid loss reached its height in the first twenty-four to thirty-six hours and then diminished. It was further demonstrated that under experimental conditions a burn of one-sixth of the body area gives a fluid loss of seventy per cent of the total blood volume in twenty-four hours. If these figures can be applied to man, it will be seen that an adult weighing sixty-five kilos and having a blood volume of 5,000 cc. would lose 3,500 cc. of fluid in the first twenty-four hours. With such a terrific loss of fluid, there is no need to look farther for the cause of blood concentration in superficial burns. This theory, then, which has been well worked out with controlled experiments, offers a conclusive and reasonable basis for at least some of the symptoms of burned patients. However, it has also been shown by the same experimenters, that by a proper forcing of fluids in the treatment of burns, it is possible to aid the body in compensating for the fluid loss, and that the blood content can be kept within normal limits. Even under these circumstances, there is still a distinct mortality in burns. This observation has been made by every one who has had any experience with burns, if the experimenter has been careful in the forcing of fluids and has kept a close study of the blood picture. It would seem, therefore, that another factor must enter into the mortality of burns. It is with this factor, and a treatment to control it, that we shall deal from now on.

There are a number of obvious features which are presented by all patients suffering from large superficial burns. Careful study of these will reveal several important points.

The first feature to be considered is the primary shock which occurs almost immediately. This seems to differ in no way from any traumatic or surgical shock and certainly occurs before any fluid loss in the form of edema of the burned area is manifested. It also occurs before increased vascularity around the burned area can be demonstrated. Consequently, it seems logical that it cannot depend on these factors. The answer to primary shock is still a debatable question and no attempt will be made to solve it at this time. It is sufficient to say that if it is combated early and adequately, with heat, rest, fluids and stimulants, it can be overcome in the majority of cases. It is our opinion that shock has little to do with the later course of the burn if the patient survives.

Once the patient has recovered from shock, he is usually fairly comfortable for the first few hours, regardless of the form of treatment which has been used on the burn. Later, under most forms of treatment, except tannic acid and the one to be described shortly, the following phenomena can be observed. The burned area becomes edematous, oozes fluid. At the same time the blood of the patient begins to show the characteristic concentration. The burn itself looks clean in the sense that there is no infection or necrosis. The patient has little pain and is not actively ill. The temperature in this period is usually normal or only slightly elevated. The pulse is usually of good quality and not markedly rapid.

Beginning about the twelfth to eighteenth hour following the burn, the patient begins to show signs of the so-called toxicity. He starts to grow stuporous, or perhaps restless. The temperature becomes elevated, the patient complains of pain; nausea and vomiting set in. Except for a slight increase in hemoglobin, leucocytosis and a moderate increase in the blood sugar and lactic acid, there is not much change in the normal blood picture, provided fluids are being forced adequately. From this time up to about the seventy-second hour, a characteristic syndrome occurs. The patient's general condition obviously grows worse. He is nauseated and cannot retain food or fluid by mouth. He grows restless and at times delirious. The face is flushed, the pulse and respiration are increased, and the temperature becomes elevated in a curve that is characterized by staircase increases, until at about the seventy-second hour it reaches a maximum of between 102½ and

105 where it is sustained for a time, plateau-like. This plateau remains constant for a number of hours, when the typical picket-fence fluctuation begins. The appearance of the burn during this period also follows a marked and characteristic change. After the first twelve hours, signs of early sepsis present themselves. The fluid over the burned areas, at first clear and odorless, becomes turbid and malodorous. The turbidity increases to the stage of frank pus and the pathognomonic odor of a burned patient is apparent. This effect also reaches its peak at about the seventy-second hour. During this time, the usual complications and sequelae such as albuminuria, extreme exhaustion, secondary anemia and delirium take place. This sequence of events during the first three days in an extensive superficial burn is remarkably constant.

Ignoring previous theories and regarding the picture speculatively, one is impressed with the fact that there is enough obvious infection present on the burned area to account for all symptoms and physical signs exhibited by the patient. It was with this in mind that Firor decided to investigate burns from a bacteriological standpoint.

Previously all the more generally accepted treatments of burns had been tried out, and compared on the same patient and on different patients. It may be stated, in passing, that the results of this investigation proved the tannic acid technique superior to others.

A review of the literature revealed the fact that no work had been done on the bacteriology of burns. Without any precedent to go on, it was decided to begin bacteriological studies on the patients as soon as they were admitted to the hospital, and follow these right through. The procedure was simple. At the time of admission, before treatment, cultures were taken of the burned area and of the fluid under the blebs, if there were any. Repeated cultures were likewise obtained at short intervals. For the first twelve hours, these areas were practically sterile, as was to be expected. A few contaminants, such as staphylococcus aureus and albus and B. coli were frequently found, but the growth was slight. After the first twelve hour period it was found that in one hundred per cent of the severely burned patients and in a large majority of the minor burns, there could be grown from repeated cultures the beta-hemolytic or the gamma streptococcus. The concentration of these organisms increased with the obvious signs of sepsis and the beginning of the toxicity of the patient, until after forty-eight to fifty-six hours pure cultures of the streptococcus could be obtained, having outgrown all the other organisms. Coincidentally, the characteristic bad effects of the burn were shown by the patient.

It seems reasonable to suppose that when a large surface of the body has been destroyed by a burn, and becomes involved with streptococcus infection, certain ill effects should result. Furthermore, the general condition of the patient and the characteristic chart of the fever would be one which coincided with that of any widespread streptococcus infection. Additional evidence has been obtained by taking blood cultures from patients after the temperature has started to swing in the picket fence curve. These cultures were positive for the invading streptococcus. Another bit of evidence in favor of our theory is that in fatal cases the hemolytic streptococcus was found in the heart-blood and in the lungs in instances of terminal pneumonias.

It was then decided that perhaps the best form of treatment would be one which aimed at killing the organisms thus sterilizing the burns. We felt that if we could show that no "toxic" symptoms occurred when the burns remained sterile, then it would be logical to assume that infection was the essential factor in the production of these "toxic" symptoms. One might suggest here that picric and tannic acids have some antiseptic properties, but they are in no way specific for the streptococcus. Their bactericidal properties are very weak, and in the case of tannic acid, once the eschar is formed, there can be no further penetration, and hence no action on the underlying infection. The coagulating properties of tannic acid which Davidson interpreted as rendering the toxic substance non-absorbable, is not so interpreted by the author, who feels that the value of tannic acid lies in the formation of an eschar over a practically sterile burn, and the sealing of the burn, which prevents fluid loss and infection. As Davidson pointed out, the general condition of the patient depended on the condition of the eschar. All large burned areas treated with tannic acid eventually become infected and it is at that time the "toxemia" develops. Three disadvantages of the tannic acid treatment are: (1) The eschar is brittle and will crack if exposed to strain. (2) The eschar is quite dense and fluid proof, so that any underlying infection will not present itself until quite extensive. (3) Minute islands of unburned epithelium such as those found in practically all third degree burns, are not preserved but are destroyed by the infection beneath the eschar.

In 1912, Churchman, working on the bactericidal and bacteriostatic properties of the aniline dyes proved conclusively that gentian violet was practically a specific for the gram-positive organisms. These experiments have been repeated by us and our results confirm his findings. In concentrations of one to one million, in test tube and agar plate, germs were not killed, but could not grow. When he increased his concentration to one to ten thousand the gram-positive organisms were killed. Churchman not

only used gentian violet experimentally in the laboratory, but also on patients. In the latter use it was not always employed superficially, but was injected in the joints and the blood stream. This dye has been used intravenously in one half of one per cent solutions for the treatment of blastomycosis and septicemias and in such quantities that the entire patient became stained, but without untoward symptoms. This last statement is offered to refute any objection which might be made as to its toxicity. In all the literature, there has been no report of gentian-violet poisoning.

Gentian-violet is a coal-tar derivative, tryphenyl-methylamine, and is slightly alkaline in its reaction. It is quite cheap, and comes either in crystalline or tablet form. The tablets should not be used, as the cohesive substance in them is quite irritating to the burned patient. It is readily soluble and an aqueous solution of one per cent can be made with great ease. The solution is sterile at all times. From this brief discussion of the properties of the dye it can be seen that it has two valuable characteristics which answer the problems offered by a burned patient; it is a specific antiseptic for the invading organism and because of its Ph reaction, together with the contained methyl radical, it reacts with the burned flesh to form a thin, light eschar, tough but flexible. This eschar when situated in the folds of the body will bend but not crack. The burn is thus sterilized and protected from further infection by an almost ideal protective eschar. The dye also produces practically instantaneous analgesia.

TECHNIQUE OF THE GENTIAN VIOLET TREATMENT

When a patient is admitted with a fresh burn, unless it is covered with oils or grease, there is no need to do any preliminary cleaning, the dye in one per cent solution being sprayed on the burned area immediately. The usual procedures to combat any existing shock are carried out. The patient is then sent to the ward and placed so that the burned area is uppermost. The burn is not covered with a dressing. The bedclothes are supported by a cradle and in the cradle a light bulb is placed. This light and the heat it generates is in no way to be confused with the dry heat treatment of burns. It is merely there to keep the patient warm. The temperature under the cradle is maintained between 84°F. and 88°F., this being the temperature found to be most comfortable for the patient. For the first few hours, gentian violet is sprayed on the burned areas every two hours. A light eschar is formed very rapidly, the wet oozing areas becoming dry and tough. The burn is thereby sealed sterilely under an impermeable cover. Further infection is thus kept out and the loss of fluids through the serous ooze eliminated. Likewise, by the time the preliminary sedative has worn off, pain has ceased. After the eschar has formed, the patient is sprayed every four to six hours during the day. Any blebs that have formed are opened and the unstained portions then presenting are sprayed. This treatment continues until healing is complete. One remarkable thing that has been noticed is that in practically none of the burns, unless actual charring has taken place, is all the epithelium destroyed and that there exist many small islands throughout the lesion which will live and spread rapidly if not destroyed by sepsis. This spread takes place unhindered under the eschar which acts as a scaffolding and which will curl up on its periphery as the healing progresses. These curls can be trimmed away to prevent the forming of any pockets for infection. If the burn is so deep and so extensive that skin grafting is going to be necessary, the eschar is allowed to remain on for about three weeks after which time it can be softened and removed by warm compresses of sterile salt solution. By that time the granulations have built up enough to graft, and, being sterile, they accept new skin readily. This is a brief description of the way in which a fresh burn is treated.

If a patient presents himself with an old burn already septic, the treatment can also be instigated with no initial clean up. The necrotic matter and pus is likewise converted into an eschar, but this eschar differs from that of a fresh burn by being irregular, in pieces, and floating. This eschar is usually removed every day and the area sprayed immediately afterward. Otherwise the treatment is the same as that described for fresh burns.

It is obvious, then, that this treatment is simple, not characterized by painful daily dressings, by having to remove the applications of the preceding day, or by manipulating and disturbing the patient. It can be carried out by the nursing staff with supervision.

The picture of a patient under this treatment is quite striking, especially when compared with patients treated with the older procedures. On inspection, whether the burn be one day or two weeks old, it is observed that the patient does not feel bad. Except for the preliminary shock, no great prostration is found. The patient is usually lying quietly in bed, in no apparent pain; he is not flushed or stuporous with fever. He is willing and able to take foods and fluids by mouth, and does not have to be carried along by the usual supportive intravenous methods. If the burn is not too extensive, the patient can actually be sitting up, taking care of himself to the extent of feeding himself and being able to walk to the bathroom. The general condition as evidenced by the chart is usually quite satisfying. The fever, if any, is moderate, and not the septic type. The laboratory work gives no indication of nephritis, or unusual changes in the blood. There is no evidence of increasing anemia since there is no blood de-

struction. The chlorides remain up and there is no increase in the non-protein nitrogen. In general, the average burned patient thus treated is not the awful spectacle that one has grown accustomed to expect. In addition, because the burn remains clean, the difficulty of handling a malodorous patient in the open ward does not arise.

It is most important to emphasize a few points. First, it must be stressed that there is no way to heal a burn and adequately care for the patient without constant supervision and interest. There is practically no burn which is so ideally situated, as to permit constant exposure to air, and so located that it is entirely free from body secretions and contamination. For example, in all large burns, certain portions will have to be undermost and these must be regarded as potentially infected. The eschar must be examined daily for softening, which may be caused by three factors; first, the secretions of the body; secondly, the underlying fat, which in severe burns becomes liquefied; and, finally, by invading infection. As soon as softening is seen, and this eschar unlike that formed by tannic acid does not conceal evidences of moisture, the soft part must be removed. This is done by picking it up with forceps and if managed carefully will not cause the patient discomfort. The surfaces thus exposed are sprayed in the usual manner.

The general care of the patient is of prime importance. This includes forcing of fluids, and a high vitamin and high caloric diet.

In brief, then, we feel that the cause of the so-called toxic symptoms seen in burned patients is secondary infection, due to the beta-hemolytic and the gamma streptococcus. This infection can be kept down to a minimum by the use of gentian violet, and if this is done, in addition to complete general care of the patient with special attention to the eschar, the prognosis in the severely burned patient is greatly improved.

SUMMARY

1. A review of the literature was made, which tended to disprove the absorption theory and the blood concentration theory as being the etiological agent in the toxicity of burns.

2. Bacteriological studies were done on a series of burned patients, and the beta-hemolytic or gamma streptococcus was found to be the invading organism in all extensive burns.

3. When a surface of the body is burned, there are no marked constitutional changes if the burns are kept free of infection.

4. It has been shown that the aniline dye, gentian-violet, is highly bactericidal against the gram-positive organisms.

5. Gentian-violet is a coagulant and an analgesic agent in burns; there is no loss of fluid from the burned area, which is sealed under the eschar. Invasion by contaminating organisms is kept down to a minimum. The patient is practically free from pain.

6. It has been shown that even in third degree burns, there are many small islands of epithelium in the hair follicles that will spread under the scaffolding of the eschar when infection is kept down, hence diminishing the necessity for skin grafting.

7. A few typical cases are reported. One of these is a third degree burn of three fifths of the body area, which lived eighty-four days and showed at autopsy none of the changes which are supposed to be associated with burns.

A REPORT OF FOUR BURNED PATIENTS TREATED IN THE BOSTON CITY HOSPITAL WITH GENTIAN VIOLET TECHNIQUE

CASE 1. The patient was a twenty-six year old white male in very good health. He was burned with scalding hot water on a boat in Boston Harbor. He was brought immediately to the Boston City Hospital. On admission he was in no shock, and immediate treatment was begun on the burns. The burns were of second and third degrees and covered

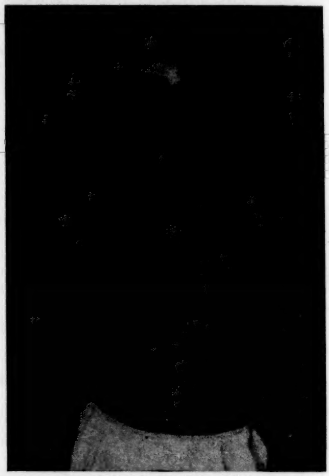
CASE 1, PLATE I.

the entire face, the upper portion of the chest, about half the neck anteriorly, and practically all of the arm surface and hands. Within twelve hours after the treatment was instigated an eschar had formed. This eschar remained dry throughout his stay in the hospital. During his stay, of nineteen days, from

CASE 1, PLATE II.

April 18, 1931 to May 6, 1931, his temperature reached 100°F. five times. The rest of the time it was under 99°F. An examination of the blood and urine revealed no abnormalities at any time during his stay. Within a week after admission the eschar

began to peel off as healing took place under it. He was discharged as completely well nineteen days after admission. The scars left by the burns were



CASE 1, PLATE III.

composed of soft, new, pink epithelium. No skin grafting was necessary. Photographs showed the patient one day after admission and on the day of discharge.

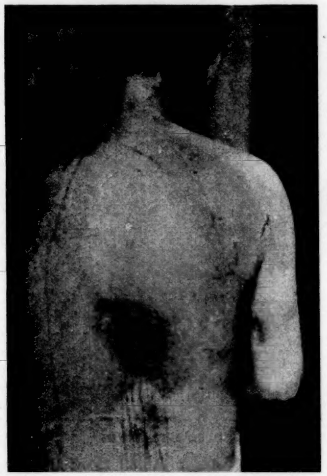
CASE 2. W. M. A forty-seven year old white male, admitted to the Boston City Hospital, March 17, 1931; discharged May 22, 1931. On the evening of March 17, 1931, the patient's clothes caught on fire when he fell asleep smoking a cigarette. He did not awaken immediately, because he was intoxicated. When he finally came to, the upper portion of his clothing had almost burned off. He was brought into the hospital in a state of mild shock. The temperature on admission was 97°F., the pulse 100, and he was in no pain. An examination revealed a third degree burn over the entire back, the back of the neck, left arm, left hand, left axilla, left chest, and about half of the right arm. He was placed immediately under a cradle lying on his abdomen and gentian violet technique was begun. The patient remained completely afebrile and free from symptoms, such as pain, discomfort, or toxicity for one week. The urine showed no casts, red cells, white cells, or albumen. At the end of the first week he became so uncomfortable lying on his abdomen that he had to be turned over on his right side. This was done only at night, as the patient was up and about during the day. It was considered more important to get the wide expanse of the back healed than to cause the patient discomfort and loss of sleep by forcing him to remain on his abdomen. Putting him on his right side, allowed the left arm to come into contact with the left chest wall. This put two burned surfaces together. However, it was deemed advisable to sacrifice the inner surface of the left arm and the left chest wall in an effort to heal the back rapidly. Within two days the eschar of the arm and chest wall had become soft and septic. Every day the soft pieces of eschar were removed, and the exposed surfaces sprayed with gentian violet. The eschar on the back began to curl up around the periphery and

CASE 2, PLATE I.

was trimmed off daily. At all times it remained dry. As fast as the primary eschar was removed the broken secondary eschar formed. The first photograph shows the patient shortly after the primary eschar of the back was removed. During this time he ran a low-grade fever, the temperature going to

CASE 2, PLATE II.

99°F. every day. At the end of five weeks the entire back had become covered with new, smooth, soft epithelium. He was then placed on his back at night, and the left arm was elevated and kept away



CASE 2, PLATE III.

from the chest wall by means of a Balkan frame. The second and third photographs show the patient three days after this procedure was started. One area in the midline in the low thoracic region of the

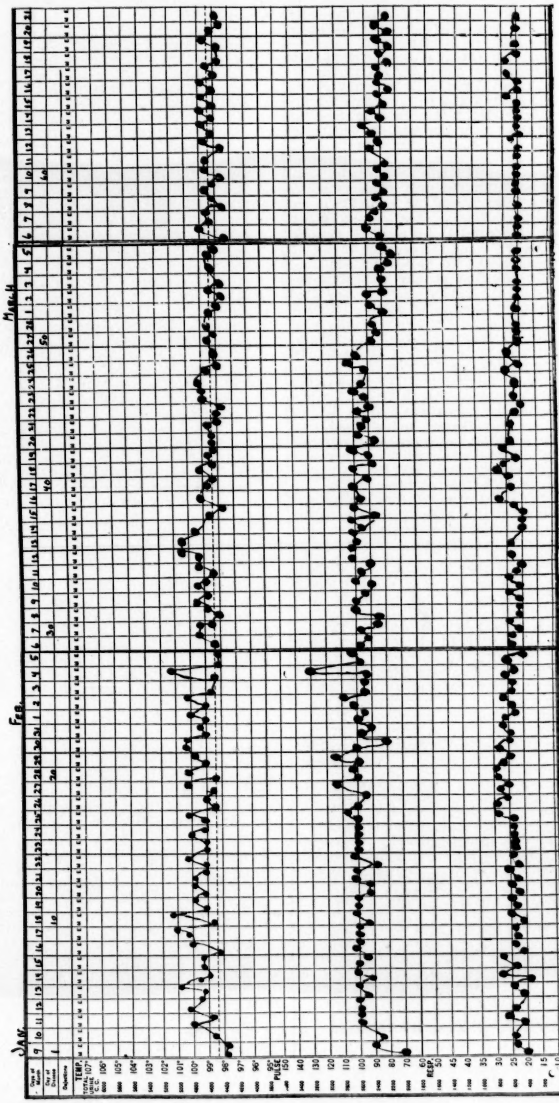
CASE 2, PLATE IV.

back broke down under the patient's weight. This area was treated with gentian violet. The burned areas on the inside of the left arm and on the left chest wall, which had been allowed to become septic, did not epithelialize. Here, gentian violet was continued until the granulations had built up enough for skin grafting. Full thickness pinch grafts were transposed from the inner aspect of the left thigh to these granulations forty-two days after admis-

CASE 2, PLATE V.

sion to the hospital. From then on healing progressed rapidly, and the patient was discharged May 22 in the condition shown in the fourth and fifth photographs. At no time during the patient's stay in the hospital were there any pathological changes discovered in the blood or the urine. He was ambulatory throughout his stay, was able to take care of himself in that he could feed himself and had lavatory privileges, and throughout his entire stay had so little pain that no sedatives were required.

CASE 3. E. A. A thirty-six year old white married female. Height, five feet, two. Weight, 212 lbs. On the evening of January 9, 1931, the patient was building a fire in a stove when her clothes became ignited. There was no one to help her extinguish the fire, and before she could do so herself, she had suffered severe burns. She was rushed immediately to the Boston City Hospital and arrived in the Accident Ward in a state of profound shock. Temperature was 97.4°F. Her pulse was very weak and thready but was only 70. She felt cold to the touch and was wet with perspiration. On examination it was found that all of her burns were third degree, and covered her left arm, left axilla, left side of the thorax, entire back, both buttocks, including the anus and vulva and the posterior portion of both thighs to the knee and the left calf. The patient was put on the dangerous list, and her relatives were told that she could not possibly live. Her shock was treated with heat, fluids, given both subcutaneously and intravenously, and stimulants in the form of caffeine and adrenalin. Within 24 hours the patient was out of her shock, temperature had returned to normal, and the pulse had risen to 90 but was of good quality. Twelve hours after her admission, gentian violet technique was begun and an eschar formed over all the burned areas within eighteen hours. Because of the burn around the



CASE 3.

anus, the bowels were kept completely closed for one week with opium. During this first week the patient's temperature rose to 100°F. on two occasions and 101°F. once. She was somewhat uncomfortable because of her position on her abdomen, but suffered no real pain. Aspirin and codeine were the only sedatives required to quiet her. The eschar remained dry, and no evidence of infection was noticed. In the second week the bowels were moved with enemata and mild laxatives. Infection spread immediately under the eschar between the buttocks. During the second week the temperature rose to 101°F. twice, and in the fourth week to 101°F. once. During the remainder of the time she ran a low-grade, swinging fever which ranged from 99°F. to 100°F. or slightly above. From the second day of her admission until discharge, she was on ward diet, and took all of her fluids by mouth. During the middle of the second week the eschar over most of the burned areas began to soften due to the burned fat beneath. As fast as any softening appeared, that portion of the eschar was removed and the spray continued. She was completely healed at the end of forty-nine days. At this time she was allowed to sit up with the result that the weight of the body on the new epithelium on the buttocks caused large cracks and fissures to appear. She had to be put back in bed and wait until these healed. She was finally discharged on March 20, 1931, completely healed. During her stay there was no change in the urine, and the only change in the blood was a leucocytosis. The smear, however, was normal. The final healing obtained was without skin grafting. There were no contractions, and the scar was the same soft, pink epithelium as observed in the previous cases. It is unfortunate that no photographs were obtained of this patient, for she is an example of a third-degree burn extending over one-third of the body area that was not a fatality.

CASE 4. R. B. A nine-year old white female, admitted to the Boston City Hospital April 4, 1931. Died June 26, 1931. The patient was playing by herself at home with some matches when her clothing caught fire. She ran into the street for help. No assistance could be obtained until all of her clothing had burned off of her with the exception of a pair of short woolen drawers. She was admitted to the hospital *in extremis*. Her temperature was 96°F. Her pulse could not be obtained anywhere. At the apex the heart sounds could be heard very faintly. The rate was about 140. An examination of the patient revealed a third-degree burn which involved the entire thorax, anterior, posterior, and lateral

CASE 4, PLATE I.

aspects, the entire circumference of the neck, the occipital region of the head, both arms from the shoulder to the mid-lower arm, both axillae, the left thigh on its lateral aspect for about one-half of its surface and both ankles. A moderately accurate estimation of the body surface and of the burned area revealed that somewhat over three-fifths of the

CASE 4, PLATE III.

body had been burned. She was immediately wrapped up in a sterile sheet and then blankets. A cradle was put over the bed. She was surrounded by hot-water bottles and put up in reverse Fowler's position. A subcutaneous infusion of normal saline was begun in the right thigh. In an hour's time intravenous saline and glucose were given. About four hours after admission the blankets and sheet were removed and the burned areas sprayed with gentian violet. This spraying was repeated every half-hour in an effort to form an eschar as rapidly as possible. At no time did she complain of pain. Six hours after admission she was definitely improved. The pulse could be obtained in the radial arteries and though rapid, the quality was not bad. One day after admission she was entirely out of her shock. Her temperature had risen from 96°F. to 102°F. The pulse rate varied between 120 and 150. Her respirations, which had been 40 on admission, had settled down to 30. From this day on until her death, eighty-four days after admission, the form of treatment consisted of gentian violet aided by all the supportive measures that could be employed. The treatment in short was simply a fight against infection all the way through. Because of the position of the burns, it was impossible to expose more than half of them to air. She was compelled to lie on her back and in order to keep this part sterile, she was placed on large pads soaked in hexylresorcinol. Repeated transfusions were given; she was put on a high-caloric and high-vitamine diet. As the eschars softened they were soaked off with sterile normal saline. She continued to run a swinging temperature of the picket-fence type which varied between 100 and 103°F. The pulse remained between 110 and 150. After a period of two months had elapsed the anterior and lateral aspects of the thorax and the anterior portions of both arms and the left thigh had formed sufficient granulations for skin grafting. It seemed at this point that recovery was possible and that as soon as the anterior portion of the body

had become covered with epithelium, the position of the patient could be changed and adequate treatment started on the back. However, on the eightieth day the child began to show marked evidence of cardiac fatigue, in spite of the fact that she had been digitalized twice to the point of vomiting in

CASE 4, PLATE IV.

an effort to slow down the pulse. From this time until her death four days later, she went steadily downhill and died of decompensation. Throughout her existence in the hospital there were no changes in the blood other than a leucocytosis. There was

CASE 4, PLATE V.

absolutely no evidence of concentration of the blood, of a change in the non-protein nitrogen or of the blood chlorides. The urine remained completely negative throughout and did not even show the so-called febrile changes. Repeated blood cultures were also negative. An autopsy was obtained and the pathological report was practically negative. She had chronic passive congestion of liver, kidneys, and spleen. There was also slight cerebral edema and congestion. All of these changes can be explained by the terminal decompensation of the heart. This case is reported as an example of how much can be accomplished in an obviously fatal burn.

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LEAD TREATMENT OF CANCER*

BY JOSEPH C. AUB, M.D.,† AND R. H. SMITHWICK, M.D.†

THE end results of the treatment of twelve cases of cancer of the breast by a combination of intravenous injections of lead orthophosphate followed by deep x-ray therapy is reported in this paper.

This work was undertaken in order to study the theory of Blair Bell[1] that lead might have a depressor effect on the rate of growth of malignant tumors, and if given intravenously might reach the cells wherever they were situated in the body.

Because of the intimate relation between lead and calcium metabolism[7], we thought that large quantities of lead could be given intravenously, in conjunction with a high calcium diet, stored in the bones, and liberated into the circulating blood as desired by combining a low calcium diet with intramuscular injections of parathormone. The tumor cells could thus be flooded with lead and acute lead poisoning avoided by reversing the process, forcing the lead back into the bones when symptoms of poisoning appeared.

We used a colloidal orthophosphate of lead solution kindly supplied to us by Dr. Henry J. Ullmann of Santa Barbara, California, which could be given in large doses with essentially no immediate reaction.

Twelve patients were selected, all suffering from carcinoma of the breast in advanced stages. In all but two, the primary lesion had been removed. Although every case had metastases to bones, or skin, or other organs, or local recurrence, they were all in relatively good condition. An effort was made to select patients whose life expectancy with deep x-ray treatment alone was estimated as long enough to decide if the added lead treatment was beneficial. A summary of these twelve cases is given in Table I.

TABLE I

	Age	Date First Symptom	Date Operation	Classification of Disease at Onset of Lead Treatment	Lead Treatment Amount (Mgm.)	Lead Treatment Duration	Lead Poisoning	Duration of Life after Lead Treatment
1.	55	13 yrs.	13 yrs.	2F	420	3 wks.	Severe	4 mos.
2.	52	4 yrs.	2 yrs.	2E	160	2 wks.	No	5 mos.
3.	41	3 mos.	None	1E	517.5	2 wks.	Severe	2½ mos.
4.	47	3½ yrs.	2½ yrs.	2E	420	3 wks.	Moderate	5 mos.
5.	39	13 mos.	1 yr.	2A	520	2 wks.	Moderate	8 mos.
6.	44	2 yrs.	2 yrs.	2E	480	2 wks.	Slight	4 mos.
7.	58	9 mos.	8 mos.	2C	500	3 wks.	Slight	14 mos.
8.	61	3½ yrs.	None	1C	557	10 days	Moderate	4 mos.
9.	56	3 yrs.	3 yrs.	2E	240	1 wk.	No	1 mo.
10.	33	1 yr.	9 mos.	2E	576	3 wks.	Slight	20 mos.
11.	55	1½ yrs.	1½ yrs.	2E	460	3 wks.	Slight	4 mos.
12.	51	1½ yrs.	7 mos.	3E	480	2 wks.	Slight	2 mos.
Averages	49	3 yrs.	2.7 yrs.		445	2 wks.		6 mos.

An average of 445 milligrams of lead was given these patients in an average period of two weeks. One patient died of an intercurrent infection four weeks after the onset of treatment. One patient could not tolerate the lead because of severe systemic reactions shortly after the injections.

When the desired amount of lead had been injected, the usual deep x-ray therapy was given. Each case received a full erythema dose of radiation at one or more areas over an average period of two weeks. Technically speaking this would mean 800-1000 r of deep therapy, K. V. P. 200,000, filtration copper ½ mm., celluloid 4 mm., distance 50 cm.

In the ten cases that received the full dose of lead, signs and symptoms of lead poisoning began to appear as early as one week after treatment was started. In no case was there any improvement in the local lesions during the period when lead alone was given. In fact, in several cases the visible lesions increased rapidly during this time. In the period of x-ray treatment, there was noticed in local lesions the usual immediate change for the better.

In eight cases, a lead line appeared within a few weeks. Six patients developed a severe secondary anemia during their hospital stay. Severe abdominal pain characteristic of lead poisoning was present in four cases. Gastro-intestinal symptoms such as loss of appetite, nau-

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sea, vomiting, or constipation, too persistent to be explained on the basis of x-ray treatment alone, were present in six cases.

These various symptoms of lead poisoning appeared with equal frequency, rapidity and severity whether the patient was on a high or a low calcium diet. The severe pain of lead poisoning was moderately well controlled by a high calcium diet and repeated intravenous injections of calcium chloride. It was necessary, however, to use frequent injections of morphine in some instances. The calcium chloride was distinctly less effective in these cases than when it is used in industrial lead poisoning. This is probably attributable to the rapid flooding of the organism with large quantities of lead. The high calcium diet, also, was not very effective, probably for the same reason. When it was most needed, the high calcium diet was sometimes not retained.

The average duration of life after the start of lead treatment was 6.2 months. Of the ten patients who received the full dose of lead, acute lead poisoning appeared to be the cause of death in two instances. Chronic lead poisoning appeared to be the principal cause of death in three cases, while five patients apparently died primarily of the disease with lead treatment acting as a contributory factor.

Extensive laboratory investigations were undertaken in regard to the lead and calcium metabolism of these patients. These were made with the purpose of determining the amount of lead retained in the body, as well as to see the influence of calcium upon its retention and circulation.

METHODS

The methods were simple. The patients were given a high calcium diet when the deposit of lead in bones was desired, but were given a low calcium diet and intramuscular injections of parathormone when mobilization with an increase of lead concentration in the blood was desired. In this way an attempt was made to keep lead circulating in the body over a prolonged period of time.

A special nurse prepared and calculated the diets and accurately collected the excreta. The periods lasted three days each and our usual metabolic technique was followed[3]. Lead was determined by Fairhall's method[4] and calcium and phosphorus by Fiske's methods[5]. The analytical data are very large but do not demonstrate anything dramatic. They are, therefore, not included here *in extenso*.

It was hoped that by means of a low calcium diet plus parathormone, the lead would be liberated from its storehouse in the bones[6] and so increase the concentration in contact with the tumor. It was also hoped that the concentration of lead in the blood stream could be reduced by changing the diet and medication to a

TABLE II

Diagnosis	Control Before Parathormone		Blood Values After One Injection of 50 Units of Parathormone						Blood Values After One Injection of 100 Units of Parathormone					
	Ca	P	Hours After Inj.	Ca	P	Hours After Inj.	Ca	P	Hours After Inj.	Ca	P	Hours After Inj.	Ca	P
	mgm. %	mgm. %		mgm. %	mgm. %		mgm. %	mgm. %		mgm. %	mgm. %		mgm. %	mgm. %
Recurrent Ca of breast	8.9	4.2	5	9.8	3.2	24	10.0	4.1	5	10.3	3.0	14	12.4*	4.3*
Ca of the esophagus	9.5	3.2	5	9.8		24	10.8		5	12.0	4.0			
Ca of the parotid	9.6		5	9.9	4.2	24	10.9	3.9	5	11.6	4.4			
Teratoma of testicle with extensive metastases	10.2	2.4	6	11.4	3.0							24	12.3	2.4
Control	10.5	4.3	6	11.1	4.2							24	13.1	4.7

*After daily injections of 100 units for three days.

high calcium intake. Unfortunately, the patients could not always retain high calcium diets, so that nearly all of them received a relatively low calcium intake. Lack of appetite, which developed during the injections of lead and during the x-ray treatments, complicated the metabolic observations so that no satisfactory conclusions can be reached in regard to the influence of treatment upon the prompt excretion of injected lead. Another difficulty which was encountered was the fact that the supply of parathormone used on these patients did not affect the blood calcium level or increase the calcium excretion. This was not due to an abnormal response by patients suffering from cancer. Subsequent observations made upon four tumor patients showed that they responded to another supply of parathormone by an elevation of blood calcium similar to that obtained in a normal individual. (See Table II.) The conclusion must, therefore, be reached that this first preparation of parathormone was inert.

The calcium excretion of patients suffering from carcinoma of the breast is practically normal. The average findings in four of the patients here reported, when compared with normal control subjects on a similar diet[7], show a slightly increased calcium excretion in the urine. This is not surprising when one considers that these patients had destructive lesions in their bones. (See Table III.)

TABLE III

AVERAGE CALCIUM EXCRETIONS IN INOPERABLE BREAST CARCINOMA

(3-day periods)

No. of Cases	Output			Intake	Balance
	Urine	Feces	Total		
	gm.	gm.	gm.	gm.	gm.
4 (carcinoma)	.50	.44	.94	.32	—.62
13 (controls)	.19	.60	.79	.33	—.46

The giving of large doses of x-ray did not affect this calcium excretion materially. It is perhaps more surprising that when large accumulations of lead were deposited in the bones there was no obvious toxic effect upon the liberation of calcium. No toxic effects were demonstrated either in changes of the calcium levels of the blood or of the urine or feces.

The lead excretion was small and a large part of the injected lead was still stored in the body when our observations ceased. Six of the cases were studied in regard to this. They received an average of 473 mg. of lead intravenously. The average duration of time in which the excretion was followed was forty-six days, during which an average of 68.6 mg. of lead was recovered. In other words, an average of more than 400 mg. was stored in the body after a month and a half of study. It is interesting that only a few milligrams were excreted even in the periods when one hundred milligrams of lead were given intravenously. This agrees with the results obtained by Kehoe and Thamann in animals[8].

The amounts of injected and retained lead in these observations are large. In previous analyses of human tissues[6] somewhat analogous amounts were found. Thus, one man dying of lead encephalopathy had only approximately three hundred milligrams stored in the body, while the maximal amount found was approximately nine hundred milligrams.

CONCLUSIONS

1. Intravenous lead treatment as given to these patients did not seem to exert any depressor effect upon the rate of growth of tumor cells.

2. By adding varying degrees of lead poisoning with the accompanying secondary anemia to the already existing disease (cancer), the duration of life of these patients was probably made shorter than it would have been with x-ray treatment alone.

3. It was difficult to control adequately the symptoms of lead poisoning by diet and calcium chloride alone. Morphine was frequently necessary to relieve the intense pain of lead colic.

4. In spite of metastases to the bones, the calcium excretion of four of these patients averaged only slightly higher than that found in normal control subjects. This calcium excretion was not influenced either by x-ray treatment or by the injections of large amounts of lead.

5. The lead which was injected averaged 473 mg. in six of these patients. An average of only 69 mg. of lead was recovered in the excreta in the next forty-six days, so that approximately 400 mg. were retained in the body.

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THE ANTIRACHITIC POTENCY OF THE MILK OF HUMAN MOTHERS FED PREVIOUSLY ON "VITAMIN D MILK" OF THE COW*

BY JOHN W. M. BUNKER, PH.D.,† ROBERT S. HARRIS, B.S.,† AND RICHARD S. EUSTIS, M.D.†

THE status of human milk in respect to its antirachitic potency is vague. In so far as its antirachitic effect upon albino rats is concerned there is considerable evidence in support of the view that it is not effective. Hess and Weinstock[1] found that 25 cc. human milk per animal per day did not protect white rats. Hess, Weinstock and Sherman[2] report that 25 cc. breast milk per animal per day for nine days did not produce healing in rachitic rats. Outhouse, Macy and Brekke[3] determined that 40 cc. human milk per rat per day produced no evidence of healing. Eufinger et al[4] state that "milk from recently delivered healthy women produced no healing of the bony rickets" (in rats), and their results are confirmed by Guggisberg[5]. Lesné and Dreyfus-See[6], and Lesné and Vagliano[7] incorporated five per cent human butter fat into the diets of rats without effect on the rachitic lesions.

In respect to infantile rickets there seems to be a general feeling that human milk, whatever its deficiencies, is superior to cows' milk in this regard but still is not reliable throughout the nursing period for the complete protection of the baby. This feeling is reflected in the general practice of enlightened pediatricians in prescribing vitamin D supplements in the diets of nursing infants, or in the diets of their mothers. The ordinary vitamin D supplements constitute in effect a special dosage in addition to the ordinary nutritional requirements, and for adults are bothersome or even distasteful.

It seems important, therefore, to determine whether the breast milk of nursing mothers can be rendered more antirachitic in some acceptable and not bothersome manner. The recent development of the Walker-Gordon Laboratories in the commercial production of vitamin D cows' milk suggests that ingestion of this milk by lactating mothers may produce the desired result.

Vitamin D cows' milk, when fed directly to infants, will cure or prevent rickets as has been demonstrated in a recent paper by Hess, Lewis, MacLeod, and Thomas[8]. It was suggested that the administration of vitamin D milk to lactating mothers might "augment the potency of breast milk which has been shown by Hess and Weinstock to be deficient in this factor." This hypothesis has been tested by us and found to be valid in the work which is herein reported.

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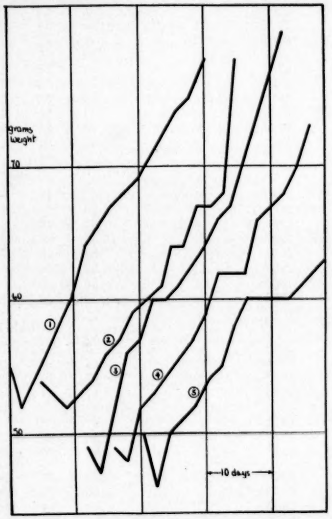
PROCEDURE

Samples of breast milk were secured from the Florence Crittenton Home in Boston from a changing group of from four to six young women between the second and fifth weeks of lactation. All were nursing their own babies. Most of them had been in the Home since a few weeks before delivery. The institutional diet was abundant and varied with eggs twice a week and green vegetables or salads daily. They took essentially 40 ounces of milk per day, of which 24 ounces was vitamin D milk (equivalent to 120 rat units). None of them took cod liver oil, viosterol, or any other antirachitic supplement.



GROWTH CURVES.

1. Average of group fed 17 cc. "vit-milk" per day.
2. Average of group fed 26 cc. control milk per day.
3. Average of group fed 15 cc. "vit-milk" per day.
4. Average of group fed 20 cc. "vit-milk" per day.
5. Average of group fed no supplement (controls).

The vitamin D milk was produced by a herd of the Walker-Gordon Company under a regimen identical with that for the herd which produced the milk used in the previous experiments of Dr. Hess[1].

Control samples of breast milk were secured from the Boston Directory, being part of a

pooled supply from 19 women living at home and nursing their own babies. They were mostly at a later period of lactation than those at the Crittenton Home. Their intake of cows' milk varied from 16 to 50 ounces per day, with an average of 27 ounces. Their diets varied widely. It is impossible to be certain of the actual foods eaten, since they kept their own records. Apparently twelve ate at least one egg per day and a moderate amount of green vegetables and salads, four more ate one or more eggs daily but no greens, and only three restricted themselves to starchy foods and milk with a little meat or fish. None took cod liver oil, viosterol, or other antirachitic supplement.

In preliminary work with Crittenton milk, the butter fat method of assay of breast milk was tried and found to be unsatisfactory. These preliminary tests suggested that about 15 cc. of whole breast milk might contain the equivalent of one rat unit. Since it was found by trial that even the young rats of a size such as

TABLE I

THE CURATIVE EFFECT OF BREAST MILKS ON INDUCED RICKETS IN ALBINO RATS

Rat number	Sex M	Sex F	Weight in grams 1st day	21st day	31st day	Degree of healing X-ray	Line test	cc./rat/day 22nd-29th day
			Controls					
2686	x		46	65	69	0	0	None
2687		x	45	53	61	0	0	"
2688	x		49	58	64	0	0	"
2689	x		49	55	62	0	0	"
2702	x		50	67	75	0	0	"
2703	x		48	66	65	0	0	"
2704	x		52	67	66	0	0	"
2705	x		50	58	64	0	0	"
2958	x		54	64	69	0	0	"
2959		x	51	56	61	0	0	"
2960	x		55	63	73	0	0	"
2961		x	55	63	64	0	0	"
			Ordinary Breast Milk					
2962	x		55	64	80	0	0	20 cc./day
2963		x	59	74	88	0	0	"
2964		x	60	73	88	0	0	"
2965	x		55	69	87	0	0	"
2966		x	52	62	74	0	0	"
2967		x	53	64	68	+	+	"
2968		x	55	73	86	0	0	"
2969	x		50	68	76	0	0	"
			"Vitaminized" Breast Milk					
2690	x		48	62	67	+	+	15 cc./day
2691	x		45	55	63	+++	+++	"
2692	x		47	62	71	+	+	"
2693	x		47	59	71	+	+	"
2970		x	52	73	82	++	++	"
2971	x		51	63	78	++	+++	"
2972		x	52	63	75	+++	++++	"
2973		x	48	66	80	+	+	"
2978		x	58	68	74	0	+	17 cc./day
2979		x	55	72	84	0	0	"
2980	x		48	58	60	++++	++++	"
2981		x	57	70	80	++	++	"
2982		x	53	65	78	+++	+++	"
2983		x	57	69	77	+++	+++	"
2984		x	62	88	100	+++	+++	"
2985	x		53	66	80	+	+	"
2694	x		49	54	66	+++	+++	20 cc./day
2695	x		47	60	72	++++	++++	"
2696	x		46	56	68	++	+	"
2697	x		48	58	72	++	++	"
2698	x		45	63	74	++	++	"
2699	x		45	55	63	++++	++++	"
2700	x		51	76	88	+++	+++	"
2701	x		50	59	67	+++	+++	"

"Vitaminized" Breast Milk=from mothers previously fed vitamin D milk.
0=severe rickets with no healing.

we use in testing can drink from 17 to 20 cc. milk per day, it was apparently feasible to assay this breast milk by feeding to the rats whole milk, and this method of feeding was pursued throughout the experiments which are herewith reported.

Animals of from 45 to 60 grams weight were caged separately, weighed each second day, and throughout the experiments maintained on the Steenbock rachitogenic diet No. 2965. At the end of 21 days these animals always showed severe rickets. For the next eight days each animal under test, except the control animals, received a fresh sample of milk pipetted into a clean jar each day, in addition to the above diet. Control animals received no milk. All animals had access to distilled water ad lib. On the thirty-first day the animals were killed, their tibiae were x-rayed and then examined by the method of the "line test". Interpretations of the line test were by the specifications of Bills[9] in which + indicates a broken line of calcification extending along the metaphysis, ++ is a narrow line but continuous, +++ indicates a broad continuous line, and ++++ complete calcification.

The essential data of these experiments and the results obtained are presented in table I.

SUMMARY

Severe rickets was the normal outcome of the diet used as is shown by the consistent findings in this regard in the entire control group of twelve animals which received no milk as a supplement to this diet.

Breast milk from mothers who have not previously been fed antirachitic supplements to a mixed diet does not heal rickets in rats when 20 cc. of milk per animal per day for eight days are fed.

Breast milk from mothers who had been fed for two weeks with 24 ounces per day of vitamin D milk as an antirachitic supplement to an otherwise adequate diet is found to contain antirachitic potency equivalent to 1 rat unit in 136 cc. of breast milk (17 cc. per day per rat for eight days). This is in spite of the fact that two out of eight animals in this run behaved in an anomalous manner and showed little or no healing (No. 2978 and No. 2979).

This breast milk when fed at the rate of 15 cc. per rat per day for eight days (120 cc. total) shows definite increase of antirachitic potency over breast milk which has not been built up by a supplement of vitamin D milk. When fed at the rate of 20 cc. per rat per day for eight days, the 160 cc. total fed per animal shows well over the equivalent of 1 rat unit.

The antirachitic potency of human breast milk can be augmented in a simple and acceptable manner by including vitamin D milk in the diet of the mother, during lactation.

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A METHOD FOR MOUNTING GROSS SPECIMENS

BY SHIELDS WARREN, M.D.*

INEXPENSIVE and compact methods for the preservation of gross specimens are greatly to be desired. A method devised by Dr. Thomas Ordway of Albany has proved so satisfactory in our hands and so many inquiries have come from those who have seen the specimens that it seems worth while to report the method of preparation, together with minor changes we ourselves have added.

The materials necessary for the mounting are a piece of glass (we have found discarded 5″ by 7″ photographic plates very satisfactory) and half a petri dish. The petri dish selected should be free from nicks and its edge ground smooth with Turkish emery and water on a slab of plate glass. The petri dish is ready for use when, if filled with water and a glass plate slid across it, the two will cohere and no air bubbles form. Once satisfactory dishes have been prepared, the actual mounting may take place.

Specimens may be preserved in any of the usual ways. We have used Kaiserling's fluid, although we find the gas method described by Robertson[1] and others more satisfactory. For the convenience of those who may not have access to Robertson's method, it is reproduced below:

A standard Whithall-Tatum museum jar, 8 x 7 5/8, is fitted with a special aluminum cover (cast to fit). In this cover are inserted two stopcocks. To one is attached a piece of glass tubing extending to the bottom of the jar and

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to the outside of this is the tube connected to the gas supply. The other stopcock is attached to a burner. As most burners have restricted openings through which the gas flows, the resistance was too great to allow the gas to bubble through the solution. Consequently a burner can be constructed by soldering an "L" shaped piece of quarter-inch copper tubing to a flat metal plate. When the gas flow is established through the fluid, this burner is ignited and the gas passing through is thus completely consumed. A chemical hood may be used instead.

Fixation: As soon as possible after the specimens are obtained, they are placed in the jar containing the following solution:

Formaldehyde	100 cc.
Sodium chloride	18 gm.
Sodium bicarbonate	10 gm.
Distilled water	1900 cc.

The solution must be changed when it becomes dirty.

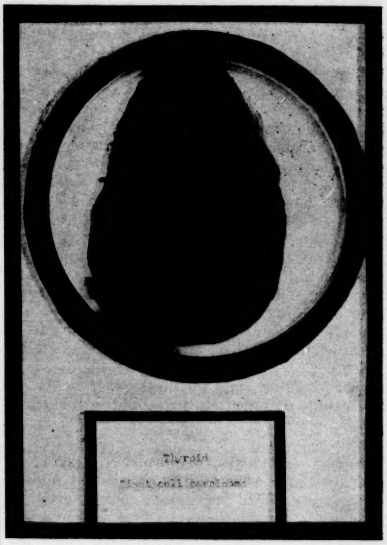
Illuminating gas is bubbled through the solution containing the specimens for about 15 minutes, after which the stopcocks are closed and the specimen allowed to remain in the gas charged solution. This is repeated each day until the specimens are fixed and the color attained is satisfactory. A week is a fair length of time. It is important that the specimens are not to be exposed to air during the process.

Mounting of Specimens: When properly fixed, the specimens are mounted in:

Cane sugar	40 gm.
Chloral hydrate	2 gm.
Distilled water	100 cc.

The one drawback to this method, which otherwise gives very faithful preservation, is the tendency to form bubbles once the specimen has been mounted. This also is a difficulty at times in other methods of mounting, so we have adopted as a routine the exhaustion of the specimens just before final mounting. An ordinary desiccator jar with a single outlet in the lid is half filled with the mounting fluid and specimens. The jar is then connected with an air pump and exhausted for a period of about twenty minutes. At the end of this time the stopcock in the entering tube is closed and the specimen is permitted to remain under partial pressure for a few hours. In this way all dissolved gas is removed from the specimen, which moreover has a slight affinity for gas so that if small bubbles happen to form in the course of mounting they are frequently absorbed by the next day.

After exhaustion, the specimen, trimmed to final form, is rapidly transferred to a fresh solution, oriented in the petri dish beneath the surface of the fluid, and a glass plate slipped over the dish and pressed into position. The mounted specimen is removed from the fluid, then carefully washed under running water to get rid of any traces of the preserving fluid, and placed inverted on a level surface to dry. As soon as dried, a coat of zinc oxide emulsified in a thin, quick-drying varnish is painted over the joints, this is permitted to dry over-



night and a second coat applied in the morning. The specimen is carefully inspected for leaks and if none develop during the following day, the zinc oxide is then rimmed over with black enamel. The final step is to bind the edges of the glass plate with lantern slide binder and to fasten with the same material a card giving the desired legend beneath the surface. The total cost of the specimen, aside from labor, is only a few cents by this method, and large numbers of specimens can be readily transported and readily stored. A carrying case for twelve or fifteen specimens need not be much larger than a microscope carrying case.

While in general this method is limited to smaller specimens, very satisfactory large mounts have been made, at times, by using panes of window glass and the larger size of glass dishes. If the specimens are not trimmed to a sufficient size to remain in place by contact with the two surfaces of the glass they may be oriented by fastening them to glass rods or by mounting on a matrix of absorbent cotton.

REFERENCE

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THE RELATION OF SMALLPOX MORBIDITY TO VACCINATION LAWS

BY S. B. WOODWARD, M.D.,* AND ROY F. FEEMSTER, M.D.*

A FEW months ago an organization opposing smallpox vaccination distributed broadcast a map of the United States in which the states having compulsory vaccination laws were colored black, those leaving the matter to local authorities were colored gray, those having no vaccination laws were left white, and those in which state laws prohibited compulsory vaccination were marked with a gold star.

This map is reproduced in Figure I. The only addition which has been made is to place in each state the total number of cases of smallpox for a ten year period 1919-1928. These figures were collected for use at the hearing on changes in the vaccination laws before a State legislative committee last year. Some interesting comparisons can be made of the figures on this map. Take for instance the black states of Pennsylvania, Kentucky and South Carolina which are surrounded largely by gray and white states and note the small number of cases in comparison to their neighbors. Observe that New Hampshire had only 86 cases while Maine and Vermont had several times that many.

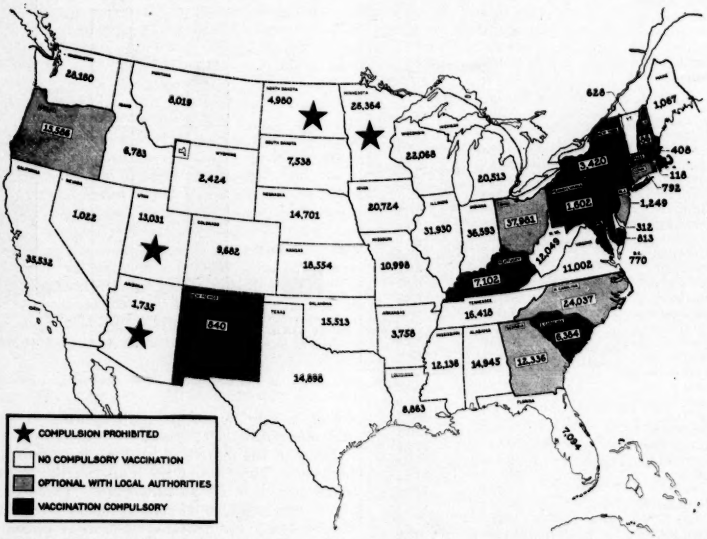

FIG. I. Cases of Smallpox 1919-1928.

A much better comparison is possible, of course, when the relative populations are taken into consideration. For this reason the number of cases and the rates per 100,000 are collected in Table I, where the states are grouped according to the type of vaccination laws.

*Woodward—President, Massachusetts Medical Society 1916-1919. Feemster—Assistant Director, Division of Communicable Diseases, Massachusetts Department of Public Health. For records and addresses of authors see "This Week's Issue," page 335.

TABLE I

TOTAL SMALLPOX CASES AND RATES PER 100,000 IN THE DIFFERENT STATES FOR 10 YEARS 1919-1928

State	Total Smallpox Cases	Rate per 100,000
States with Compulsory Vaccination		
Massachusetts	408	1.0
Pennsylvania	1,602	1.7
Rhode Island	118	1.8
New Hampshire	86	1.9
New York	3,420	3.0
Maryland	813	5.3
District of Columbia	770	16.7
New Mexico	840	21.5
Kentucky	7,102	28.3
South Carolina	6,384	37.4
States with Local Option		
New Jersey	1,249	3.5
Connecticut	792	5.3
Georgia	12,336	42.6
Ohio	37,981	62.2
North Carolina	24,037	84.0
Oregon	15,586	179.5
States with No Compulsory Laws		
Delaware	312	13.6
Maine	1,067	13.6
Vermont	628	17.7
Arkansas	3,758	20.9
Texas	14,898	28.4
Missouri	19,998	31.4
Illinois	31,930	45.4
Louisiana	8,863	45.5
Virginia	11,002	46.5
Michigan	20,513	48.3
Florida	7,094	58.4
Alabama	14,945	60.1
Mississippi	12,136	63.7
Tennessee	16,418	66.5
Oklahoma	15,513	70.3
West Virginia	12,049	75.2
California	35,532	78.2
Wisconsin	22,068	79.2
Iowa	20,724	85.2
Colorado	9,682	89.9
Kansas	18,554	101.9
Nebraska	14,701	110.1
South Dakota	7,538	113.5
Wyoming	2,424	115.9
Indiana	36,593	118.5
Nevada	1,022	122.0
Montana	8,019	147.5
Idaho	6,783	155.0
Washington	28,160	193.0
States Prohibiting Compulsion		
Arizona	1,735	45.1
North Dakota	4,980	75.4
Minnesota	26,364	106.3
Utah	13,031	272.0

The differences are even more striking, however, when we compare the rates of the groups, as shown in Figure II. The bars in this chart show in an unmistakable way that vaccination controls smallpox. The *black* states had only

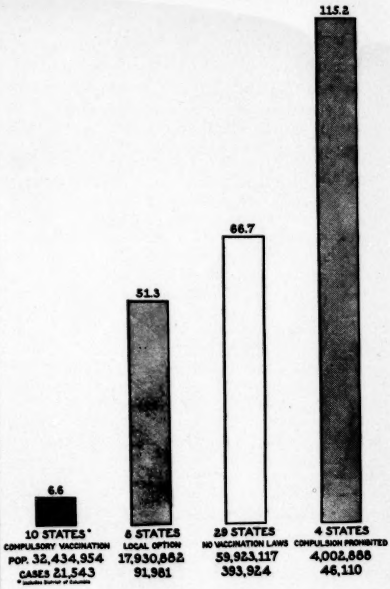

FIG. II. Cases of Smallpox per 100,000 Population 1919-1928.

6.6 cases per 100,000 population, while the *gold star* states had 115.2 cases.

Some of the figures given here were published last year[1], but neither the map nor the bar diagram was printed at that time. These charts have been used so effectively in driving home the necessity for vaccination that it seems but right to put them where they are available to those who may want to use them.

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PERSISTENTLY POSITIVE BLOOD REACTIONS IN TREATED SYPHILIS*

BY WILLIAM D. WHEELER, M.D.‡

PERSISTENTLY positive Wassermann reactions in treated syphilis have created a puzzling problem for some time, and the question has become even more perplexing with the advent of more specific and more sensitive blood tests for syphilis.

There is no doubt now that the Wassermann test on the blood, while definitely of certain value, never was a complete criterion for estimating the cure of syphilitic infection. The large number of relapses described by various observers bear witness to the fallibility of the test[1, 2]. Monorecidive chancres, late secondary lesions, neurorecurrence, organic involvement, and infection of marital partners have been reported after years of negative Wassermann reactions[3, 4, 5], and syphilitic offspring have been born to parents apparently cured of syphilis, as indicated by the reaction of the blood. Asymptomatic relapses were also numerous in which the serum reactions which had been negative became positive again but in which there was no return of clinical symptoms[6-17].

The more recently developed tests, such as the Kolmer, Kahn, and later the Hinton test, are described by many observers in this country and abroad as possessing greater sensitiveness than the Wassermann test. With the aid of these tests, particularly the second modification of the Hinton test†, syphilitic infections have been discovered, which were previously undiscovered because the Wassermann reaction was negative[18, 19, 20]. Furthermore, because of the sensitiveness of these tests it has been at least suspected that many treated patients who had been pronounced cured, because of absence of symptoms and negative Wassermann reactions, were in reality not cured. However, the question of cure is far from completely answered, for the problem now concerns not only the Wassermann-fast cases but also the Hinton-fast cases, and due to the very sensitiveness of the Hinton test, Hinton-fast cases have become more numerous.

Beinhauer mentioned a number of definitions of a Wassermann-fast case[21]. He selected Schamberg's definition as the best: "A Wassermann-fast reaction is one that remains positive or is uninfluenced by long-continued treatment, which is usually able in most cases to reverse a positive Wassermann." This applies just as properly to a Hinton-fast case. Three distinct groups of such cases can be recognized in syphilology: (1) cases of early syphilis in which positive blood reactions recur following several negative reactions; this is usually due to irregular or insufficient treatment and is easily corrected by renewal of treatment; (2) cases of late syphilis in which a persistently positive reaction is accompanied by clinically recognized organic involvement; most of these cases do not yield to any form or length of treatment, and (3) truly asymptomatic cases in which no clinical evidence of syphilis can be found; the only indicator of infection is a persistently positive reaction of the blood. It is with the two last groups that this paper is concerned; two hundred such cases were selected for study from the Department of Dermatology and Syphilology of The Boston Dispensary.

ETIOLOGY

The significance of the persistence of positive reactions is as yet little understood. The general tendency is to regard it as evidence of a focus of treponemata hidden somewhere in the body. Whether the treponemata in such cases are pathogenic remains to be proved. Stokes and others[22] who see in this phenomenon an indication of menacing presence of syphilis have advised much treatment. Wile[2], on the other hand, considered the situation not at all serious and advised very little, if any, treatment. He pointed to the futility of trying to obtain a negative blood reaction in congenital syphilis or neurosyphilis. Both Thom[23] and Belding[24] advanced the theory that these resistant reactions are caused by special strains of the *Treponema pallidum*. They expressed the belief that arsenic-resisting strains exist which may be transmitted to others. Tobias[1] spoke of the probable biochemical fate of arsenic in the body. According to this theory, certain ferments in the blood prevent the transformation of the arsenic into an active spirillicide. Tobias also mentioned two other theories. According to one theory, described by Marshall, certain biological substances are formed in the blood of the patient by the treponemata, and these substances persist long after the death of the organism. According to the other theory, advanced by Warthin, the patient is a treponema-carrier. Warthin described a perfect parasitism whereby the treponemata become encapsulated and harmless, causing only a non-specific reaction in the tissues. The antisyphilitic drugs are incapable of penetrating these capsules. Jameson[25] gave coincident foci of nonsyphilitic diseases as contributing factors in the persistence of a positive

*From the Department of Dermatology and Syphilology and the Department of Clinical Research of The Boston Dispensary. Read at a meeting of the Clinical Staff of The Boston Dispensary, November 1, 1932.

†A third modification of the Hinton test has recently appeared.

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reaction. Almost all syphilologists agree that the most important factor is the presence of syphilitic nests in such organs as the heart, lungs, aorta, spleen, liver, bones, and brain[20], although the actual mechanism by which these persistent reactions are produced still remains a mystery.

MATERIAL STUDIED

In common with similar clinics, the Department of Dermatology and Syphilology of The Boston Dispensary, with its large group of syphilitic patients, has a number of Hinton-fast and Wassermann-fast cases. During the five years preceding this study, the number of cases of active syphilis under treatment averaged 900. This number increased in the year of this study to 1200. Of these, about 300 have been found to be Hinton-fast cases, and to date 200 of these have been studied.

The investigation was made in such a way as to discover, if possible, the cause of the persistently positive blood reaction. A thorough search was made for hidden syphilitic foci and for nonsyphilitic ailments. For the latter purpose the aid of the Medical Department of the Dispensary was enlisted.

The following data were charted for each case: (1) sex, age at the time of the first examination, occupation, and habits; (2) age at the time of infection and date of infection; (3) duration of infection; (4) diagnosis and date at which diagnosis was made; (5) serum reactions; (6) previous treatment (intravenous medication, intramuscular medication, other treatment, time of first treatment, and remarks as to irregularity of treatment); (7) statement as to whether there was involvement of heart and aorta, spleen and liver, or nervous system; (8) coincident nonsyphilitic ailments; (9) untoward reactions to treatment; (10) new treatment, and (11) any change in serologic reactions.

It seemed necessary to determine at first one important factor: Were these persistent cases truly asymptomatic or did the patients have any complaints, signs, or symptoms? Each of the 200 patients was asked the following questions: "Does anything hurt you or distress you? Have you any pain, ache, or discomfort at any time through the day, night, week, or month?" Barely five per cent had no complaints to report. All the others had some distressing symptoms. In some cases the complaints when investigated proved to be apparently fanciful, or at least no clinical evidence could be found to substantiate the symptoms. The majority, however, had definite pathologic conditions. Of the 200 patients, 192 were found to have definite syphilitic involvement of one or more organs. Sixty-seven patients had coincident nonsyphilitic ailments, sixty of which were gonorrheal. There were among these sixty-seven cases, four of diabetes, two of pulmonary tuberculosis, eight of definitely diseased tonsils, forty-five of various stages of decay of teeth and pyorrhea, numerous cases of dermatosis including erythema induratum, lichen planus, lupus vulgaris, lupus erythematosus and dermatitis herpetiformis, two cases of epithelioma of the face, a number of cases of uterine retroversion, and a large number of cases of dermatomycosis. Eight of the 200 patients were without any symptoms and without demonstrable clinical evidence of syphilis. These were the only truly asymptomatic cases.

One hundred and twelve of the patients were found to be working at hard labor, fifty-one were alcoholics, one patient was a cocaine addict, forty patients suffered from overeating, and 108 had moderate habits.

The duration of the disease was studied next, with special reference to the lapse of time between infection and diagnosis. It was found that in 147 of the cases a diagnosis of syphilis was not made until from two to forty years after onset.

The serum reactions of the 200 patients at the beginning of this study were as follows: One hundred and sixty-six had persistently positive Wassermann reactions and the entire 200 had persistently positive Hinton reactions. Seventy patients had received either insufficient or irregular treatment, consisting of short courses of arsenic, dosage 0.3 to 0.5 gm., with long intervals between treatments, ranging from a few months to five years. In forty-four cases treatment consisted only of arsenic used rarely and in small amounts, the dosage never exceeding 0.2 gm. on account of untoward reactions. Much bismuth was used in this group of cases. Untoward reactions in fifty-eight of these cases were responsible for the insufficient treatment; forty-four of the reactions were about equally divided between nitritoid and jaundice. The remaining reactions were arsenical collapse (two cases), optic atrophy (one case), arsenical dermatitis (three cases), gluteal hematoma (one case), renal involvement (one case), hysteria (two cases), and malingering (four cases).

Among the 192 patients with syphilitic organic involvement, cardiovascular lesions predominated, there being sixty-five in all. Of these, there were four cases of aneurism, five of aortitis, eleven of arteriosclerosis, and forty-five of aortic heart disease and hypertrophy. Neurosyphilis was next in frequency, the number of patients being twenty-four, of whom twenty were alcoholics. Meningovascular lesions were present in twelve of the twenty-four cases, tabes was present in three cases, paresis in two cases, and a positive spinal fluid only in seven cases. There were twenty-two patients with splenohepatic lesions, six of whom had lesions of the spleen, and two of whom had syphilitic jaundice later. Congenital syphilis was present in thirteen cases; keratitis was noted in three of these cases, and other stigmata were noted in ten cases. Syphilis of bones was

present in four cases, including affections of the sternum, both tibiae, and Charcot joints. Syphilis of the eyes was present in two cases, manifested as retinitis and beginning optic atrophy. In two cases the kidneys were affected; the syphilitic element was not definitely determined; there were no urinary casts, and no pus; blood and albumen, which were cleared up by eighteen injections of arsphenamine, were present. Various combinations of syphilitic involvement existed in sixty cases. About twenty of the alcoholics had neural involvement, and almost all of these were subject to gastric reactions after arsenical treatment.

It is apparent that organic syphilitic involvement, wrong habits and coincident nonsyphilitic foci may be logically suspected as being contributory factors at least in the persistence of positive serum reactions. Careful search and inquiry usually will reveal both symptoms and organic involvement in most cases. Again it should be emphasized that but eight of the 200 patients had no complaints or any clinical evidence of organic involvement, the persistently positive serum reaction being the only suggestion of syphilis.

TREATMENT

Since this investigation has been carried out over a period of barely a year, conclusions would not be justifiable in regard to the efficiency of the various methods of treatment used. Brief mention of the procedures employed will suffice at this time.

First an attempt was made to clear up all nonsyphilitic foci of disease. Next the 200 patients were divided into groups for the purpose of antisyphilitic treatment. The largest group, consisting of 100 patients, was given intensive treatment with bismuth, either large doses once a week intramuscularly or three smaller ones thrice weekly intravenously. These were for the most part patients subject to arsenical reactions, some with mild neural involvement and some with inaccessible veins. Negative serum reactions resulted in two of these cases, and the reactions have remained negative. To another group of eighteen patients oral medication with metaphen was prescribed, one teaspoonful three times a day, a rather large dose. There was no improvement in serum reactions after three months of medication, but some patients have reported improvement in general health, which may have been purely psychic. A third group of thirty patients, noted for previous irregularity in attendance, was treated three times a week with small doses of some arsenical preparation intravenously. No change occurred in serum reactions after three months of treatment. The patients then became very irregular in their attendance. A group of fifteen patients, subject to arsphenamine and neo-arsphenamine reactions, was treated with silver arsphenamine once a week, bringing the dose up to 0.3 gm. After twenty-four injections, three of these patients gave negative blood reactions. One patient with syphilis of the sternum and another with involvement of both tibiae were treated with inhalations of ethyl iodide three times a week, as advocated by Swartz, Blumgart and Altschule[27]. There was definite improvement in both cases. Of thirteen congenital syphilitics treated with bismarsen, five gave a negative blood reaction after fifteen injections. Three of the five patients soon gave a positive blood reaction after a rest of six weeks. There was symptomatic improvement in all of these congenital syphilitics.

Fourteen patients were given stovarsol tablets to be taken three times daily, each pill containing four grains, corresponding to 0.1 gm. of arsphenamine. These patients previously had had much intramuscular treatment and hardly any arsenicals on account of untoward reactions. In one case of this group a negative blood reaction developed which stayed negative even after a prolonged period of rest from treatment. One patient, who was subject to arsenical reactions, had similar reactions from stovarsol so that it had to be stopped. In another case jaundice developed. The remaining twelve patients reported marked symptomatic improvement. Treatment with stovarsol was carried on for about four months, the dosage being diminished to half the ordinary amount at times. Stovarsol, the story of its development and its value merit further study. It has its definite place in the treatment of syphilis, particularly in infants and children, for patients with inaccessible veins, and for persons such as travelers who cannot visit a clinic regularly. While most workers, in describing this drug, admit its value, they all agree that it is a makeshift remedy, has certain dangers, and cannot replace intravenous therapy[28-31].

The eight truly asymptomatic cases did not have any treatment.

Thirty other patients were studied as controls. Treatment was started in the primary or early secondary stage, and was persisted in regularly for two years or slightly longer. The Hinton reactions in these cases remained negative after periods of rest from treatment.

SUMMARY

(1) Two hundred patients with persistently positive serum reactions were studied and tabulated.

(2) Only eight of the 200 patients presented neither lesions nor symptoms.

(3) A large majority (147) of the patients started treatment two to forty years after the date of infection.

(4) Seventy patients had irregular or insufficient treatment, particularly lacking in arsenicals; these patients demonstrated that untreated or insufficiently treated syphilis is a serious

menace, causing grave damage to the nervous, vascular and visceral systems.

(5) Although modern treatment is not finally ideal, and has certain disadvantages, it is nevertheless of essential value until further progress is made.

(6) It is reasonable to suppose from the observations that were made in this study that organic syphilitic involvement is in most instances the cause of persistently positive serum reactions and that such reactions usually mean uncured syphilis.

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PROVISIONAL SUMMARY OF MORTALITY STATISTICS: 1931

The Bureau of the Census announces that in the United States death registration area, in the calendar year 1931, there were 1,322,587 deaths, or a death rate of 1107.5 per 100,000 population, as compared with 1,343,356 deaths, or a rate of 1133.1 in 1930.

The five leading causes of death in 1931 were the same as in 1930, namely, diseases of the heart, cancer, nephritis, cerebral hemorrhage, and the pneumonias, and in each of the years these five causes were responsible for slightly over 50 per cent of the total number of deaths which were reported in the registration area.—*Department of Commerce, Bureau of the Census, Washington.*

HEALTH CONDITIONS IN MOSCOW

According to reports received by the *New York Times*, the health of the children in Moscow is most satisfactory, for the writer declares that there is no city of four million inhabitants in the world in which the children are healthier and happier than in the Soviet Capital. Nor is there any appreciable evidence of adult privation, although there is much anxiety among the people. One reason for the present well-being of the children is alleged to be that their diet consists very largely of farinaceous food with meat three times a week only, in the majority of cases. Under the system in vogue the children get a balanced meal in the schools either free of charge or at small cost if the parents can afford it.

One peculiarity of this eastern city is that there are no slums in Moscow like those of the western part of Europe. New buildings are generally erected on the outskirts of the city. The suburbs appear very much like overgrown villages and have no concentrated population. The general construction of Moscow seems to be an improvement over many of the large cities of the world, inasmuch as there is abundant opportunity for the children to play in open spaces. What is more surprising, no family seems to be too poor to afford adequate clothing. The rules with reference to attire appear to be "wear as little as possible in summer and as much as possible in winter."

It has been observed that the Russians as a class are extremely careful of their children. The reaction from these conditions is shown in the mental attitude of the children, who are constantly singing and laughing and have a definite interest in family life.

Medical records and vital statistics bear out the surface impression of juvenile Moscow's healthiness. The death rate among infants during the last year touched the record low figure of 118 per thousand as compared with 256 in 1913.

VERMONT STATE MEDICAL SOCIETY

NON-TUBERCULOUS PULMONARY SUPPURATION*

BY EDWARD D. CHURCHILL, M.D.†

Mr. President and Members of the Vermont State Medical Society:

IN speaking on non-tuberculous suppuration of the lung I shall confine my remarks to lung abscess, particularly in its acute stages, bronchiectasis and empyema with bronchial fistula.

The problem of the etiology of lung abscess following operation has occupied the attention of the experimental surgeon for the last ten years and there has been an active dispute between two schools in regard to causative factors. Time does not permit a detailed discussion of the two theories: (1) aspiration of infective material via the bronchial route and (2) blood borne infected thrombus.

The greater number of thoracic surgeons believe that aspiration is the chief cause in the cases that we meet clinically. It is very comforting to the nose and throat specialist who is so unfortunate as to have a patient develop lung abscess following tonsillectomy to hold to the idea that it was of hematogenous origin, viz., that a septic thrombus formed in the veins of the tonsillar fossa and was transported to the lung by the blood stream. I take issue with this view and do not believe that he should find comfort in that thought, for in my opinion the abscess developed following the aspiration of infected material from the pharynx either during or within the first few hours following operation. For this reason the operation of tonsillectomy must be regarded as of major importance, carrying with it a definite mortality and it must be surrounded by greater safeguards, particularly in adults. Acute lung abscesses occur with greatest frequency following operations on the nose and throat, particularly tonsillectomy. Tooth extraction and other operations on the jaw are also, frequently contributing causes.

Certain abscesses develop in the lung following pneumonia. Classical lobar pneumonia is almost never followed by abscess formation. Bronchopneumonia, particularly of a septic type with mixed bacterial flora, is a frequent cause of its production. There are in addition a number of abscesses that seem to develop without known cause. The patient may have a slight cold or respiratory infection followed by the spontaneous development of a large abscess in the lung.

A fourth important cause of lung abscess is the necrosis and breaking down of a malignant tumor of the lung. Malignancy of the lung, particularly in its later stages, may masquerade as a lung abscess. This type is particularly to be found in older patients with the so-called spontaneous type of abscess.

We cannot consider the diagnosis of lung abscess in detail but the clinical picture is entirely familiar to you. Foul sputum, perhaps hemorrhages, fever, leucocytosis, harassing productive cough, rapid loss of weight and strength combine to produce a characteristic picture which differs markedly from the usual case of pulmonary tuberculosis. The physical signs of lung abscess are far from being characteristic as signs of cavitation almost never exist. Certainly in the acute stages the cavernous type of respiration so commonly heard in advanced fibroid phthisis is almost never heard in lung abscess due to the fact that the lung tissue surrounding the cavity is infiltrated with a pneumonic lesion in contrast to the hard, sclerosed tissue that surrounds the cavity of pulmonary tuberculosis. The x-ray picture may offer a ready diagnosis or may be very confusing. A fluid level is of great help in making the diagnosis by x-ray but in certain cases may be lacking due to the fact that the cavity has inadequate drainage and never empties itself completely enough to form a fluid level.

The problem of treating lung abscess is most important. Once the diagnosis is established, it depends first of all upon x-ray localization and the course of treatment must be guided by frequent x-ray films taken during the progress of the disease.

The detailed treatment of lung abscess was then discussed and illustrated by lantern slides showing the anatomy of the lung and the importance of correct localization of the disease.

Bronchiectasis was then discussed and the progress made in the treatment of this condition by surgical methods pointed out. Eight cases of lobectomy in patients suffering with bronchiectasis confined to one lobe of the lung were reported with a mortality in this series of zero.

The diagnosis of empyema with a bronchial fistula was discussed and illustrated by case histories and lantern slides.

*Read at the Annual Meeting of the Vermont State Medical Society, at Burlington, October 6, 1932.

†Churchill—Chief of the West Surgical Service, Massachusetts General Hospital. For record and address of author see "This Week's Issue," page 335.

MISCELLANY

VERMONT DEPARTMENT OF PUBLIC HEALTH

DECEMBER, 1932

The incidence of communicable diseases for the month of December is as follows: chicken pox 307, diphtheria 9, measles 7, mumps 216, scarlet fever 86, smallpox 1, typhoid fever 1, tuberculosis 9 and whooping cough 98.

A total of 1,455 examinations was made by the Laboratory of Hygiene. They were as follows:

225	Examinations	For Diphtheria Bacilli
15	"	For Widal Reaction of Typhoid Fever
196	"	For Tubercle Bacilli
404	"	For Evidence of Syphilis
132	"	For Gonococci in Pus
38	"	Of Water, Chemical and Bacteriological
236	"	Of Water, Bacteriological
60	"	Of Milk, Market
60	"	Of Milk, Submitted for Microscopical only
2	"	Of foods
3	"	For the Courts, autopsies
9	"	For the Courts, Miscellaneous
1	"	To Complete Death Returns
1	"	Of Animal Heads for Evidence of Rabies
73	"	Miscellaneous

The Division of Venereal Diseases has received reports of thirty-eight cases of gonorrhea and twenty-two cases of syphilis. Two hundred and twenty-six Wassermann outfits and 96 slides have been distributed from this office.

The nurses of the Division of After-care for Poliomyelitis made 66 home visits during December, four doctors were called on, one patient admitted to the Children's Hospital, two patients were admitted to the Audubon Hospital and three patients discharged from hospitals. Twenty pieces of apparatus were fitted, five pieces of apparatus were altered and eleven orthopedic corrections made to shoes. The vocational worker of this Division reported sales amounting to $127.95.

Nine towns were visited during December by the State Advisory Nurse, assisting physicians in the examination of school children, giving talks to various organizations and meeting with Home Demonstration Agents.

DR. MERRITT H. EDDY REACHES HIS ONE HUNDREDTH BIRTHDAY

Believed to be the oldest practicing doctor in America, Dr. Merritt H. Eddy passed his one hundredth birthday at his home in Middlebury, January 25, 1933. While unable to be out he enjoyed the day by reason of the felicitations of his many friends and exchanged comments on the active years of his life, especially enjoying a delegation from the University of Vermont Medical College, the Chi Psi Fraternity of Middlebury College, listening to college songs by the present undergraduate student body and communicating with his relatives on the telephone. He received telegrams and letters, one letter being from President Hoover.

Dr. Eddy graduated from Middlebury College with the class of 1860 and from the Medical College of Burlington five years later and is the oldest living graduate of these institutions.

He has been especially regarded as the typical family doctor and has witnessed the making of medical history perhaps more than any other living physician. He is especially interested in the changes in medical practice since Civil War days and has observed the progress of surgery from its crude earlier stages to a fine art. He notes that medical chemistry has become a field in itself and that well-qualified general practitioners become interested in the very many specialized branches of the art.

Although very much pleased with the opportunity to confer with many people, Dr. Eddy maintained an attitude of conservatism with reference to questions propounded by various newspapers and press services. He did not refuse interviews with those with whom he felt acquainted, but he wished little or no heed paid by the outside world to the long service which he has rendered to humanity. He showed the breadth of his interest in the affairs of life by discoursing on the changes taking place in the business world as well as in the practice of medicine.

Dr. Eddy is a member of the Methodist Church and a Mason.

His son, Dr. Stanton S. Eddy, is now practicing in Middlebury and his two grandsons, Dr. Stanton, Jr., and Maxon Eddy, are in Boston. The latter is a medical student.

CHITTENDEN COUNTY MEDICAL SOCIETY

At the annual meeting in September Dr. A. L. Fogg was elected president, Dr. W. G. Townsend, vice-president, Dr. Sam Sparhawk, secretary and treasurer.

At the November meeting the society had an honor conferred in the privilege of listening to an address by one of the former graduates of the University of Vermont, Dr. K. J. Tillotson, who spoke on Psychiatry in Medicine.

The date of the annual meeting has been changed to May in order to obviate any further difficulties in elections to the House of Delegates.

The consensus of opinion was to have the meetings bi-monthly in the future, as most of the members have to attend the various hospital meetings each month so that they can adhere to the tenets of the American College of Surgeons, thus relieving the members of attendance on too many meetings.

NORTHEASTERN COUNTY MEDICAL SOCIETY

The January meeting of the Northeastern County Medical Society was held at the Newport House, Newport, Vt., Wednesday evening, January 25. At this time three former members were reinstated to active membership, Dr. J. M. Blake, Barton; Dr. J. F. Blanchard, Newport, and Dr. L. N. Piette, Newport.

The guest speaker of the evening was Dr. J. A. Nutter of Montreal who spoke on "Chronic Arthritis." His address was illustrated by many lantern slides.

ADDISON COUNTY MEDICAL SOCIETY

A meeting of the Addison County Medical Society was held at the Porter Hospital in Middlebury, January 4, 1933. Dr. Richard S. O'Connell of Middlebury, formerly of Teaneck, N. J., was voted a member. There was a general discussion of group and illness insurance.

It was voted that the Secretary write a letter of congratulation to Dr. Merritt H. Eddy, long a member of this society, on the occasion of his one hundredth birthday, the twenty-fifth of January.

MORE INTEREST IN THE STUDY OF PUBLIC HEALTH

That there is a marked increase in the number of Americans studying at the School of Public Health at Harvard was cited recently by Dean David L. Edsall in his annual report. He stated that in the past the demand for well-trained men in public health has been limited by "undesirable political influence in the appointments of health officers in many parts of the country" and gave the assurance that these conditions are slowly improving. Contrary to previous years, when the students have been very largely foreigners sent here by foundations or governments, the School entered this year with twenty-eight students and of the total twenty are Americans. This increase, while not great in total numbers, is regarded as sufficiently adequate to meet the need for public health officials in this country.

One may now expect a moderate but continuing supply of well-trained men who will exert a very marked influence upon the administration of public health policies in this country.

The Harvard School of Public Health has coöperated with governmental, semi-public and other organizations. During the past year the United States Navy sent several officers to the School to carry out investigations with reference to the atmospheric changes in submarine work. Such coöperation is regarded by Dean Edsall as of especial value in dealing with general scientific problems.

POSITIONS IN PUBLIC HEALTH SERVICE

According to information disseminated by Congress, there are soon to be opportunities for a considerable number of physicians in the public health service. This department of the Government is one hundred and thirty-four years old and since 1889 the personnel has been on a commissioned basis comparable to that of the Army and Navy. There are eight Assistant Surgeons General stationed in Washington. Their relative Army rank is that of colonel with the highest base pay $4,000 a year to which there is added $2,000 longevity pay, $820.40 rental allowance and $379.60 for subsistence, a total of $7,200 a year.

A surgeon stationed in Boston, whose corresponding rating in the Army would be that of a major, draws base pay of $3,500; longevity allowance, $1,750; rental, $1,296, and subsistence, $569.40, a total of $7,115.40.

There are about three hundred and eighty such positions throughout the country and a few for stations in foreign countries. Each appointee to these positions will be by presidential selection. These positions are restricted to physicians and surgeons. They are of great interest to the medical profession.

THE QUESTION OF DOCTORS AS LEGISLATORS

Dr. Bernard Sachs, recently elected President of the New York Academy of Medicine, in his inaugural address, urged the members of his profession to take more active part in public life. He feels that doctors cannot be indifferent to the basic issues of honesty and efficiency in government, but says that "there is one important reason for not filling up all the Legislatures with medical men" because in course of time any considerable predominance of physicians in legislative bodies would lead to a gradual development of the usual defects of political activities. Overemphasis of one particular type of mind would follow, just as would happen if the governmental departments were filled with engineers, architects, painters, or even sculptors or editorial writers, and the same arguments would apply in that people in groups of sufficient size to influence legislators would be very likely to develop policies too definitely connected with their particular problems. This viewpoint seems to be very logical, because it is well to recognize the fact that making a good statesman usually means spoiling a good engineer or physician.

CASE RECORDS
of the
MASSACHUSETTS GENERAL HOSPITAL

ANTE MORTEM AND POST MORTEM RECORDS AS USED IN WEEKLY CLINICAL-PATHOLOGIC EXERCISES

EDITED BY RICHARD C. CABOT, M.D.
F. M. PAINTER, A.B., ASSISTANT EDITOR

CASE 19061

EIGHT MONTHS' FOUL SPUTUM

SURGICAL DEPARTMENT

A nineteen year old American girl was sent to the hospital from a State sanatorium. The chief complaint was cough of seventeen months' duration with foul sputum for eight months.

She had had two or three sore throats a year until she was twelve, and a peritonsillar abscess at ten. Since she was twelve she had had no sore throats. Nineteen months before entry she had tonsillectomy and adenoidectomy under ether. She did not regain strength after the operation, and two months and a half later began to have morning cough with half an ounce of thick green sputum daily, never foul. She had sweating every night for five months. After a month she began to tire easily. She raised three teaspoonfuls of bright red blood, and from that time had blood streaked sputum about once a week. A month later, fifteen months before entry, she began to have pain in the right chest, worse on breathing, persisting six months. Eleven months before entry she had to go to bed because of weakness and malaise. A month later her temperature was 101°. Three weeks after this she was sent to a sanatorium, where she had remained until entry. Eight months before entry her sputum was foul for the first time, making her vomit daily. Three months before entry she was having five sputum cupfuls a day. The amount gradually decreased to three cupfuls. Three weeks before entry all her symptoms improved and she began to gain in weight and appetite. She had previously lost 12 pounds in nine months.

The past history is negative except for possible diphtheria in infancy and headaches on exertion for the past six months.

Physical examination showed a fairly well nourished girl with a flushed face, looking ill. One left cervical gland was enlarged. The left side of the chest was prominent and larger than the right. There was marked lag in expansion of the right side, which hardly moved at all. The trachea was slightly displaced to the right. The right lung showed flatness with bronchial breathing, increased tactile fremitus and spoken voice throughout, but no egophony. There was an area two inches in diameter below the right clavicle where the breathing was amphoric and the percussion note had a metallic quality, possibly coin sound. There were coarse moist râles there and occasional fine moist râles over the rest of the chest. The heart was normal except for a scratchy systolic murmur at the base. The abdomen was negative. The fingers were moderately clubbed. Rectal examination was negative. The pupils and reflexes were normal.

The urine was not remarkable. Blood examination before operation showed 4,540,000 reds, 11,700 to 4,800 white cells, 46 to 65 per cent polynuclears, hemoglobin 60 per cent. The smear showed moderate achromia, slight variation in size and shape, increased number of platelets. A Hinton test was negative. A tuberculin test, 1 to 20,000, was negative; another, 1 to 1000, was positive. One stool the day after admission showed a large amount of gross blood. Eleven examinations during the following days were negative.

On x-ray examination the entire right lung field showed diminished density sufficiently intense to obliterate the outline of the right border of the heart. Within the dullness in the upper third there were numerous irregular areas of decreased density. The heart and trachea were considerably displaced to the right. The entire left lung field was clear. Three days later the area of diminished density in the right lung field was perhaps a little larger. No fluid levels could be distinguished. Four days later the areas of decreased density were still visualized. No definite fluid levels were shown either in the anteroposterior view or with the patient on her side. Five days later the area of mottled dullness was slightly less dense than at the previous examinations and there was now a suggestion of a fluid level. The patient was put on postural drainage twice a day with production of half an ounce to an ounce each time.

Eighteen days after entry operation was done. The following day the patient was fairly comfortable. She raised large quantities of blood stained sputum. There was profuse foul drainage from the wound. She coughed a great deal. The temperature swung from 101.5° to 102°, once to 103.2°, during the first five days. During the next five it swung at a lower level. Then it reached normal. On the eleventh day she complained of a tingling sensation in the right fourth and fifth fingers. On the twentieth day she complained of a little pain in the right shoulder. There was still profuse drainage from the wound. On the twenty-seventh day 5 cubic centimeters of lipiodol was allowed to spread throughout the bronchi. X-ray examination showed the upper part of the lower lobe of the left lung fairly well injected and apparently normal. A medial bronchus was also visualized and appeared normal. There was no injection of the right lung. On the forty-ninth day an-

other lipiodol injection was done. X-ray showed the right lung field apparently much the same as it had been at the last examination except that several dilated bronchi were visualized in the midlung field. Throughout the left lung field there was soft mottled dullness which was distributed from the apex to the base but did not become confluent. It appeared to conform with the bronchial and alveolar markings, probably representing lipiodol. No dilated bronchi were demonstrated on the left side.

On the fifty-second day the right phrenic nerve was crushed under local anesthesia.

On the fifty-ninth day fluoroscopy showed the appearance in the chest to be much the same as before. There was no evidence of collapse of the right lung other than displacement of the heart towards the right. The position and appearance of the left lung remained the same.

On the seventieth day a third operation was done. There was profuse drainage after it and the patient was quite toxic, as had been expected. She was given postural drainage in the shock position. There was no sign of bleeding. She raised sputum and had fits of coughing on the slightest motion. On the seventy-fifth day during a fit of laughter she had pain in the right chest, coughed up large clots of blood, became cyanotic and died.

Clinical Discussion

Dr. Richard C. Cabot: The only institutions that I know of called sanatoria in this state are for tuberculosis. She came, then, from an institution for tuberculosis.

In tuberculosis the sputum is sometimes foul, but not often. Our attention is turned towards abscess by the foulness of that sputum. This is a perfectly typical history of a lung abscess following the commonest cause in this part of the world, that is a tonsil operation. So far as our indications go it seems to be on the right; that is, the pain is on the right. We have nothing else. And the history shows a period of improvement which we often do see in lung abscess when it is draining freely.

"The trachea was slightly displaced to the right," that is towards the side where we so far have reason to expect trouble.

The auscultatory signs of abscess of the lung are anything or nothing. I do not know any physical sign that you may not have with abscess of the lung. You may have the signs as of fluid when there is fluid present or when there is none, from great thickening of the pleura. You may have the signs of solidification so that you get the impression of pneumonia, or you may get the signs of cavity, as apparently they did in this case. On the other hand a small abscess or even a number of small abscesses may cause no physical signs whatever, or only a few râles from the bronchitis about them. So anything between the very marked signs that we have here and the minimal signs mentioned may occur. All that we regularly find when we listen to the chest in a case which we suspect to be abscess because of its history is something wrong, usually something wrong in that chest!

I am interested in the displacement of the trachea. I do not remember that much in relation to abscess of the lung. But one can see that if there were retraction of the lung or plugging of the bronchus which might be there in connection with the abscess, there would be drawing over of the trachea with the rest of the mediastinum towards that side.

I have assumed that the diagnosis is abscess and nothing else. We have to consider foreign body or malignant disease, either of which might cause the same signs and history, although we should not expect them to follow an operation in this way. Of course malignant disease might come on after an operation, or a foreign body might have got in after an operation.

This is just such a slight secondary anemia, with moderate leukocytosis at one time, as we should expect in abscess of the lung. The leukocyte counts go up and down ordinarily as the abscess drains better or worse.

Of course with a concentration as great as that a large number of people would have a tuberculin reaction from some obsolete focus. It has no diagnostic value here.

Gross blood in the stools I think one hardly can account for by abscess of the lung. One has to look elsewhere for the reason of that. "Eleven examinations during the following days were negative." I do not think we can make much of it then. We may suppose it was a hemorrhage from piles. Some hemorrhoids bleed very intermittently.

We will see the x-ray plates taken before operation.

Dr. George W. Holmes: The films cover a period which began in March and finished in June. The most noticeable thing is the complete absence of normal lung markings throughout the right side of the chest, with the displacement of the heart and mediastinal contents towards the affected side. The shadow is more marked towards the base, where it obliterates the outline of the diaphragm. Towards the apex it becomes mottled, and within the mottled portion are bright areas suggestive of cavities. This film was taken with the patient lying on her back and with a Bucky diaphragm to bring out the dense portion of the lung in the hope that we might be able to show its mottled area better. I do not think these plates give much added information except that the process remains about the same for a period of a month.

Then we have the films taken after the injection of lipiodol. We can see the lipiodol along the trachea and the bifurcation of the bronchus

with the lipiodol streaking down into the divisions of the bronchi and the alveoli of the lung. In this area we see that the bronchi stop rather abruptly and the lipiodol seems to be in small globules rather than in the fine distribution seen in other parts of the lung. In other words, the bronchi on this side are not so well shown as on the other side. Any process as dense as this, occupying the whole lung, must be due to fluid or to destruction of the lung or both. Fluid within the pleural spaces without collapse or destruction of the lung would tend to push the heart and trachea in the opposite direction. If there is a large amount of fluid present we must assume that the lung is collapsed or destroyed to a considerable extent and that it is not being inflated in the usual way. A shadow as dense as this, obliterating the whole outline of the diaphragm, almost invariably means some fluid; but we should not expect that the major process was fluid in the pleural space, but that it was perhaps secondary to the process in the lung and that we were dealing with a rather extensive destructive process in the lung itself.

DR. CABOT: How much does the x-ray help to distinguish between a tuberculous and a nontuberculous process?

DR. HOLMES: I think that with tuberculosis as extensive as this would have to be we should have some changes in the other lung. That is not always so, but usually is with a process as extensive as this. Then it is rather unusual for tuberculosis to produce as much displacement of the heart and trachea as we see here without a large cavity at the apex. So I should say the evidence we have here would be in favor of a very extensive destructive process in the lung with some involvement of the pleurae and a small amount of fluid, with some evidence against tuberculosis rather than in favor of it but not ruling it out by any means.

DR. CABOT: Is there anything to show a single drainable cavity?

DR. HOLMES: No, I should say the evidence we have is against its being a single cavity. The cavities are probably multiple, small and lobulated.

DR. CABOT: Is there anything that suggests a fluid level there? They refer to it in the text.

DR. HOLMES: It is very difficult for me to point one out. There are a number of plates and it is quite possible that in some of them there is something suggestive, but there is nothing definite here. Of course a cavity in which there is a fluid level is a little less likely to be a tuberculous than an abscess cavity. Sometimes if we take films with the patient lying on his side we get a suggestion of fluid level, or we can demonstrate that the shape of the shadows changes with the changed position of the patient.

DR. CABOT: Now we are coming to the operation and we ought to commit ourselves on a preoperative diagnosis before we ask Dr. Churchill to tell us about the operation itself. The striking thing is how much more there is than just the abscess that one would have suspected from the history. I believe there is very little sound lung on the right side. If you say, what is the matter with that lung, I think you have to say it is chronic pneumonitis, partly fibrosed inflammation of the lung presumably around a number of abscesses. But it is not clear from the evidence so far that there is any single abscess much larger than the rest. The question of fluid we cannot settle if Dr. Holmes cannot. In many of these cases you do not have fluid. In practically all you have so much thickening of the pleura that you have the appearance of fluid. I have no doubt there was much thickening here, that the pleura was obliterated practically everywhere. So I should expect that the operation would show, so far as it has the opportunity to do so, a chronic pneumonic process through most of the lung in addition to one or more, probably many, abscesses, and that there has been some shrinkage of the lung to account for the deviation of the trachea in that direction. I see no evidence of foreign body or of malignant disease, and I should say that while either of these things would cause most of this picture, the history is probably reliable in a nineteen year old girl against malignant disease and against foreign body.

DR. EDWARD D. CHURCHILL: I think I am responsible for emphasizing the importance of recognizing a deviation of the trachea particularly in surgical cases. Probably because of being a surgeon, I find that I cannot percuss the borders of the heart with any great degree of accuracy. In fact, I am a little skeptical at times regarding the performances of my medical colleagues, as nearly any one of them can be deceived if a case of dextrocardia is surreptitiously slipped under his fingers. In a case like this, with marked changes in one side of the chest and perhaps compensatory changes in the other, I hesitate to interpret the findings of percussion irrespective of their accuracy. Hence it is very important in relation to the presence or absence of fluid to determine which way the trachea is deviated.

In judging the clinical picture and pathology in this case before operation I think we have two things to help us, chiefly the physical signs and to a less extent the x-ray. Usually the reverse is true in dealing with lung abscess. We had here distinct signs of cavitation below the right clavicle, and it was this cavity which we proposed to drain. There was also present an extensive destruction of the remainder of the lung, probably secondary to the one large cavity.

PREOPERATIVE DIAGNOSIS

Lung abscess.

FIRST OPERATION

Local novocain. Sections of the third and fourth ribs were removed in the midaxillary line. The pleura was very thick and adherent. With the electrocautery a button was removed from the pleura and underlying lung, opening into a cavernous upper lobe abscess. The wall was smooth but there was accumulated cheesy pus in the crypt of the ramifications of the cavity. A tube was inserted for drainage.

PATHOLOGIC REPORT

Chronic pneumonitis.
Bronchiectasis.

FURTHER DISCUSSION

DR. CABOT: I should like to ask a little about the indications for operation here, and the prognosis which Dr. Churchill would make both before and after it. How do you decide in such a case whether it is going to be well to operate or not?

DR. CHURCHILL: In this particular case and in similar cases we are faced with a desperate situation,—a patient with a chronic lung abscess of seventeen months' duration with extension of the disease to the rest of the lung, but the process being unilateral. It is at least conceivable that we could help such a patient by several operations spaced at intervals, based on combining drainage and collapse procedures.

DR. CABOT: That is, she would have no chance at all without operation, and there is evidence to believe that she would have some chance, though not a very brilliant one, with operation. After you had made your incision did you feel any different from the way you did before the operation?

DR. CHURCHILL: I felt that the patient would probably have some reduction in the amount of sputum following drainage of the cavity and it would then be safer to proceed with thoracoplasty or cautery pneumonectomy.

DR. CABOT: After the first five days I suppose everybody was beginning to say, We have done a good job; we have relieved her of part of her burden and perhaps she can bear the rest.

I do not know enough to interpret the tingling sensation in the fingers. Of course, in any lung abscess we are always thinking of the possibility of brain embolism, and I suppose that the man who wrote down that finding about the fingers was wondering if she had an embolus so minute that it affected only that part. The brain should be in our minds all the time in a case of this sort.

The lipiodol "was allowed to spread". That is the favorite medical slang. The lipiodol wanted to get in; you just gave it permission and it went. They did not get any to spread into the right side. Does that mean that the bronchus was closed?

DR. HOLMES: Some part of it probably. This is the film that showed that. We can see that as compared with the other side it is blocked more or less there from pressure on the bronchus.

DR. CABOT: We have to suppose, I understand, on the basis of this examination, that there was little if any entrance to the main bronchus on that side. On the forty-ninth day there is a question of a general bronchiectasis; infection on the left side from the right is what is in their minds.

Have you anything more to tell us about the crushing of the phrenic nerve, Dr. Churchill?

DR. CHURCHILL: It was an attempt to reduce the cough as another step in the surgical attack in this case.

Her sputum diminished somewhat following the drainage of this large cavity, but she was still raising two or three boxes a day of foul purulent sputum and was draining from the side. So we proceeded with this operation.

PREOPERATIVE DIAGNOSIS

Lung abscess, right.

THIRD OPERATION

Avertin, gas-oxygen. Anterior axillary line incision curving backwards towards the angle of the scapula and cutting through the border of the latissimus dorsi. Sections of three ribs were then removed downward and backward from the draining sinus. With the electrocautery the intercostal bundles and parietal pleura were removed. The lung was completely adherent. With the electrocautery lung tissue was then removed, excavating an area the size of a teacup. Below, the diaphragm was exposed in an area about two centimeters in diameter. No division was found recognizable as the middle lobe. We were however very definitely working in the lower lobe. The lung tissue was studded with bronchiectatic abscesses and was completely fibrosed and atelectatic.

PATHOLOGIC REPORT

Bronchiectasis.
Chronic pneumonitis.

FURTHER DISCUSSION

DR. CHURCHILL: We found the lower lobe to be completely adherent. No middle lobe was recognized. There was very little hemorrhage connected with this procedure. Following the partial cautery-pneumonectomy, as this operation would be called, this area of excavation in the lung was packed very firmly with gauze.

DR. CABOT: I think if one cannot be allowed to laugh after such an operation it would be a serious element in making one wonder whether it is worth while to have it.

With such admirable opportunities for biopsy as we have in this case, I do not see how Dr. Mallory is going to add much about the lung. The question for us is, I think, to say what else there will be in this patient outside of that right chest cavity. Here is chronic suppuration of the type often associated with amyloid.

DR. TRACY B. MALLORY: Our examination was limited to the thorax.

DR. CABOT: How about the heart? Have we any reason to suppose this heart is going to be abnormal? I do not see that we have. Of course a heart can become hypertrophied, on the right side especially, from a chronic fibrosed process, ordinarily in both lungs. I do not know of a proved case of cardiac hypertrophy from disease in one lung alone. So I see no reason to suppose any disease in the heart or any considerable disease in the left lung.

How much harm can lipiodol do in the lung?

DR. HOLMES: None.

DR. CABOT: So we have no reason to suppose that there was any injury in the left lung or that infection had taken place there through the other bronchus. Dr. Mallory ought to describe a lung full of holes, bronchiectasis, fibrous tissues, an adherent pleura and nothing else.

CLINICAL DIAGNOSES (FROM HOSPITAL RECORD)

Lung abscess.
Bronchiectasis.
Massive hemorrhage.

DR. RICHARD C. CABOT'S DIAGNOSES

Chronic pneumonitis (peribronchial).
Bronchiectatic abscesses.
Chronic pleuritis with thickening.

ANATOMIC DIAGNOSES

1. *Primary disease.*

(Pulmonary abscesses, right.)
Bronchiectasis, right.

2. *Secondary or terminal lesions.*

Operative wounds: thoracotomy, cautery pneumonectomy.
Hypertrophic pulmonary osteoarthropathy.

3. *Historical landmarks.*

Chronic fibrous pleuritis, right.

PATHOLOGIC DISCUSSION

DR. MALLORY: Dr. Cabot seems to have covered it completely; I think I have not much to add. The left lung appeared entirely normal except for a certain amount of compensatory emphysema, and the entire right lung, that is all portions of it in the pneumonectomy, were filled with numerous bronchiectatic cavities and a diffuse fibrosis in between these cavities. There was no tissue on that side possibly capable of any respiratory function. The heart was a little smaller than normal, if anything.

A STUDENT: What killed her?

DR. CABOT: Was there evidence of a fresh hemorrhage, Dr. Mallory?

DR. MALLORY: We didn't notice very much.

DR. CHURCHILL: Clinically she died of hemorrhage. She was comfortable when the house officer saw her and before he had left the ward she had died.

DR. CABOT: But if she had died of hemorrhage we ought to see it, inside of the body or out of it.

DR. CHURCHILL: I think she coughed up most of the blood. We removed the pack and put in another one.

DR. MALLORY: There was a little hemorrhagic fluid in the bronchi.

A STUDENT: You don't think she died of suffocation?

DR. MALLORY: Once in a while in a case of this kind we see aspiration of blood from one side of the lung into the lung on the other side. That was not present in this case.

DR. CABOT: I think the question is an important one. People can lose an enormous amount of blood without dying. I think we have to say she was so near the edge of death that a relatively small bleeding was enough to kill her. Of course the main cause of death is sepsis, I take it.

DR. MALLORY: I think pulmonary hemorrhage accounts for fully thirty per cent of lung abscess deaths. In the group of cases we have in this hospital it is much commoner for fatal pulmonary hemorrhage to occur from abscess than from pulmonary tuberculosis. It is very common in abscess, and very often terminal and fatal.

DR. CABOT: But think how much blood people lose by stomach or by rectum sometimes and live along perfectly well. It is surprising. Could one have taken out the whole lung?

DR. CHURCHILL: We were not planning on that, although it might have been desirable. What we wished to do was to give adequate drainage to the numerous cavities and then by rib resection produce collapse of the remaining cavities. It is a discouraging type of case. In one just like this a year or two ago we attempted to produce collapse before we tried the drainage, and the patient died of pneumonia.

DR. CABOT: I don't see why the lipiodol did not come into the right lung more.

DR. MALLORY: There is no mention of bronchiolar stenosis. Exudate might prevent it from going in, and you would not want to use much pressure.

DR. MAURICE FREMONT-SMITH: Doesn't lipiodol slip in because of the increase of the size of the thorax, as it were, and wouldn't the absence of expansion account for the failure of the lipiodol to go in?

DR. CHURCHILL: Possibly.

DR. HOLMES: It is a fact that when there is an abscess of the lung the lipiodol tends to form in the bronchi around the periphery of the abscess. Unless the abscess is draining into the bronchi there would be no chance for the lipiodol to go into the abscess cavity. But we have been able to demonstrate the area of inflammation in the lungs from the fact that the lipiodol failed to go into that area.

DR. CABOT: In relation to the physical signs, I think the only possible way is to pay no attention to the physical signs in abscess of the lung, because the different elements that we have, the thick pleura, the pulmonary solidification and the cavities are such that I do not think any man can figure it out. I think the people who examine most of these cases do not try.

A STUDENT: What was the infectious agent in the abscesses?

DR. MALLORY: I do not think we made any attempt to investigate it.

DR. CABOT: I think generally the bacteriology of such cases is wholly unproductive, there are so many different things there and we cannot tell which one is doing most harm. One of the things that seem most stupid in textbooks is the way they have a separate section on gangrene of the lung and one on abscess of the lung. So far as I know they are the same thing in signs, causes and results. There is only a slight difference in bacteriology. So far as I see they ought to be combined into a single disease.

CASE 19062

LOWER ABDOMINAL DISCOMFORT AND A MASS IN A MAN OF FIFTY-EIGHT

SURGICAL DEPARTMENT

A native born stationary fireman fifty-eight years old entered complaining of weakness, anorexia and lower abdominal discomfort of fourteen months' duration.

With the onset of the symptoms he had a marked change in his bowel habits from constipation of many years to loose watery movements, never more than three a day, always in the morning and about one hour apart. The abdominal discomfort was constant, not related to food or relieved by soda or defecation. Seven months before entry a barium enema in the Outpatient Department showed no definite evidence of disease. After taking a prescription of bromide, nux vomica and gentian compound the diarrhea stopped and he felt better for two months. Then he began to have weakness and dyspnea, to perspire freely and to develop a severe constant pain localized at McBurney's point, worse on standing after sitting for some time and relieved only by lying on the right side. For two months before entry he had been passing light colored stools. During the past year he had become nervous, depressed and irritable. He thought he had lost 20 pounds in the past seven months.

He gave a past history of "typhoid pneumonia" thirty-six years before entry, a year later pleurisy. Thirty-five years before entry he had chancre and gonorrhea. Twenty-six years before entry he had erysipelas and sixteen years before entry influenzal pneumonia. The same year he was operated upon for a blocked right ureter and was quickly cured.

The family history is not contributory.

On physical examination he was a fairly well nourished man with no abnormalities except for a slightly tender, freely movable, not definitely fluctuant mass the size of a baseball over McBurney's point.

The urine was normal. Blood examination showed 5,060,000 red cells, 11,800 white cells, 83 per cent polynuclears, hemoglobin 65 per cent, moderate achromia, slight variation in size. The stool showed a very strongly positive guaiac.

Two days after entry operation was done. The patient was in fairly good condition the following day, with a satisfactory chart. On the second day he vomited, but otherwise continued to do well. On the morning of the fourth day he was thought to be making excellent progress. A little later he suddenly became cyanotic, unconscious and pulseless, and within a few minutes died.

CLINICAL DISCUSSION

DR. MAURICE FREMONT-SMITH: Of course the first thing we think of in any man of fifty-eight who has a marked change in his bowel habits is cancer. Sometimes patients say that they are relieved; they find that whereas they have been constipated for a long period of time, now their bowels move easily. The doctor should be suspicious of such a change taking place in that period of life. The fact that he had his movements all in the morning and none in the rest of the day might be an argument against malignancy, and make us feel there was some irritative process which was stimulated by the first meal of the day and which was less effective later. You often find patients who are smoking too much who have diarrhea; two or three movements early in the day and nothing else afterwards. Probably it is the effect of the nicotine on an intestinal tract which is particularly susceptible to irritation owing to the fact that there is less food in it.

The x-ray showed no disease, and would be pretty good evidence that there was no malignancy.

He "developed a severe constant pain localized at McBurney's point, worse on standing after sitting for some time and relieved only by lying on the right side." That would lead us to think again that this was something outside

the intestinal tract, that the bromides had quieted down his intestinal tract sufficiently so that this irritation, whatever it was, did not cause frequent movements. It would also suggest the possibility at least of a mechanical something which was relieved by lying on his right side and which when he lay on his left side pressed or pulled on the peritoneum in such a way as to give him pain.

"For two months before entry he had been passing light colored stools." Unless he was jaundiced or unless he had sprue, I do not know what that means. We have nothing here to show that he was jaundiced. Perhaps he was not digesting his fats; perhaps he had something the matter with his pancreas.

I do not feel that I have any answer to the problem as the result of reading this history. Of course we know that there is no such thing as "typhoid pneumonia." We should have talked about that a good deal ten years ago. Often with typhoid there is a bronchitis or even a pneumonia, but it is typhoid fever and not typhoid pneumonia.

I wonder what had blocked that right ureter. He was fortunate to be "quickly cured".

There is nothing in the past history that I can think of as bearing on the present illness. What can we consider? In the first place the whole history is too long for an appendiceal abscess. An appendiceal abscess is sometimes walled off from the rest of the peritoneal cavity, so that I can conceive of moving it. I think I have seen one that could be moved, but it would never be described as "easily movable". What else? I cannot think of malignancy in the intestinal tract that would be freely movable. Malignancy connected with the colon would be inside, not outside the colon.

A STUDENT: How about a cyst?

DR. FREMONT-SMITH: What sort of cysts could we consider? A pancreatic cyst is of course out of the question. That is too high and never freely movable. A mesenteric cyst I think might be possible, one that pressed on the intestine and caused no change in the x-ray. Could he have had an echinococcus cyst? Of course that is merely a possibility, a guess. Is there anything that could possibly be connected in any way with the old operation on the ureter? I cannot see anything.

The white cell count is just a little bit above what we should accept as normal. The white cell count is said to vary one hundred per cent in the same individual. If it is 6,000 at one time it might be 12,000 at another and still be physiological. On the other hand, when we find a leukocyte count of over 10,000 I think we should consider it presumably increased. It is not often much higher than 10,000 in acute appendicitis. It may be lower, may be normal, four, five, or six thousand. The point is, when we say 12,000 it is probably a little high.

Nothing that I have thought of excepting malignancy would show a strongly positive guaiac unless he had some interference with the blood supply to the intestines, in which case he certainly would have symptoms, not of mild pain on change of motion but of acute obstruction, as in a mesenteric thrombosis or a twisted intestine from hernia. That may cause blood and a positive guaiac. But we thought we had eliminated the inside of the intestinal tract successfully, and now we find something which can be explained only by a lesion inside the intestinal tract.

We have to decide what they operated for. They operated for a mass, and the mass was causing pain. I am entirely unable to make a preoperative diagnosis, and I should like to know what the House Staff thought.

DR. RICHARD C. CABOT: I do not see why it should not be a cancer of the cecum. So far as I see it, your chief reason against it is that it is freely movable. I think I have seen cancer of the cecum quite movable.

DR. FREMONT-SMITH: And seven months after the onset the x-ray was normal; but x-rays can be wrong.

DR. GEORGE W. HOLMES: We make a certain number of errors in the examination of the colon, and with almost everything else in favor of cancer I should assume that the x-rays were wrong in this case.

DR. FREMONT-SMITH: Would you expect him to go as long as this with cancer of the cecum without more irritation or more obstructive symptoms?

DR. CABOT: I think cancer at that point is terribly slow sometimes. I should suppose they had made a preoperative diagnosis at that point of cancer of the cecum.

DR. EDWARD D. CHURCHILL: I know nothing about the case, but should agree with Dr. Cabot that the evidence indicated cancer of the cecum.

PREOPERATIVE DIAGNOSIS

Carcinoma of the cecum.

OPERATION

Spinal novocain, gas oxygen and ether. Through a right paramedian incision a large tumor was found involving the cecum and adherent to the lateral peritoneum. There was no evidence of metastatic glands in the mesentery distant from the tumor. The tumor was removed by a right colectomy after division of the terminal ileum and the right half of the transverse colon. The ends of the small and large bowel were turned in and closed by suture and the continuity of the intestinal tract reëstablished by a side-to-side anastomosis of the ileum and the transverse colon. The abdomen was closed without drainage.

PATHOLOGIC REPORT

Adenocarcinoma, grade II.
Healing appendix.
No metastases in lymph nodes.

FURTHER DISCUSSION

DR. FREMONT-SMITH: The death was probably from pulmonary embolus.

CLINICAL DIAGNOSES (FROM HOSPITAL RECORD)

Carcinoma of the cecum.
Pulmonary embolism.

DR. MAURICE FREMONT-SMITH DIAGNOSES

Cyst of the mesentery?
Pulmonary embolus.

ANATOMIC DIAGNOSES

1. *Primary disease.*

(Colloid carcinoma of the cecum with metastasis to the liver.)

2. *Secondary or terminal lesions.*

Operative wound, ileostomy.
Pulmonary embolism, right.
Pulmonary congestion.

3. *Historical landmarks.*

Chronic fibrous pleuritis, marked.
Tuberculosis of the spleen with calcification.
Tuberculosis of the mesenteric glands, healed.

PATHOLOGIC DISCUSSION

DR. TRACY B. MALLORY: At autopsy we found a pulmonary embolus. The right pulmonary artery was completely filled with a clot which was dark red and fairly elastic, and at first we thought it looked like a postmortem clot, but on examining it carefully we found that the branchings of it did not at all correspond to the branchings of the pulmonary artery. The fourth day after operation is about as early as one ever sees a fatal pulmonary embolus, so that of course this was a much fresher clot than the average fatal embolus that we see in the lungs.

There was very little else of any significance in the body except three fairly good sized metastases in the liver.

DR. FREMONT-SMITH: Adenocarcinoma?

DR. CABOT: Did you see the specimen, Dr. Mallory?

DR. MALLORY: It was a rather typical colloid carcinoma. Those grow with quite variable rates of speed but often are fairly slow in growth.

DR. CABOT: Was there any obstruction?

DR. MALLORY: I have not the complete report and I cannot answer. The metastases were in the liver, none in the regional lymph nodes. There were a few calcified nodes in the mesentery, but none that showed tumor.

DR. CHURCHILL: How early after operation have you found fatal pulmonary embolism?

DR. MALLORY: I think, as I have said, this is about the earliest. I have never seen it in three days except where there was good reason to suppose a thrombus had been present preceding the operation. In several genito-urinary cases I have seen it on the second or third day, and in those cases we have found a thrombus of the periprostatic plexus of veins. That is a very common place for thrombosis in non-operative genito-urinary cases. We had for instance a short while ago a fatal pulmonary embolus undoubtedly from that plexus in a case where the only operation was the crushing of a bladder stone, not a cutting operation at all. They simply put an instrument into the bladder and crushed the stone, and in a few days he had a fatal pulmonary embolus. In the majority of postoperative cases I think thrombosis does not occur until after the operation, and I do not believe one sees emboli much before the fourth day. Does that agree with your observation?

DR. CHURCHILL: Yes.

A STUDENT: Was this a metastasis by the blood stream to the liver?

DR. MALLORY: I should imagine so, via the portal vein.

DR. FREMONT-SMITH: That is unusual.

DR. MALLORY: Theoretically it is unusual, but in practice it happens all the time, rather particularly from the intestinal tract, I should say.

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THE COMMISSION ON MEDICAL EDUCATION

THE Association of American Medical Colleges organized in 1925 the Commission on Medical Education "to make a study of the educational principles involved in medical education and licensure, and to make suggestions which would bring them into more satisfactory relationships with the newer conceptions and methods of university education, on the one hand, and with the needs of present-day society, on the other". The Commission, under the chairmanship of President Lowell of Harvard University, was composed of a number of nationally known physicians and educators. Dr. Willard C. Rappleye was Director of the Study.

The report of the Commission, made public late in 1932, is of increased interest coming as it does in a period of great national anxiety when every man is lacking that sense of security so necessary for well-being, and coming practically simultaneously with the report of the Committee on the Costs of Medical Care. The two reports, indeed, deal with such closely interrelated subjects that they can be considered as striking at the same general problem from two slightly different angles. The first chapter of the report, in fact, on public aspects of medicine, is based largely on the economic aspects of medical care, making due acknowledgment of the fact that the present widespread propaganda regarding the economic aspects has focussed attention upon the present forms and costs, rather than upon a plan which will insure services of high quality. It is pointed out that the total expenditures are a small fraction of the national income and insignificant when compared with the vital values which the services aim to protect.

Certain fundamental principles have been established as a result of the studies made, and certain underlying faults in our present scheme of medical organization, or disorganization, have been brought to light. The statement is almost axiomatic that an understanding of the medical needs of the population and the qualifications which the physician should possess in order to deal most effectively with those needs should be the basis of medical training, rather than the forms and subdivision of medical practice, which are determined to a considerable extent by economic and other factors.

The study has revealed that there are more physicians in the United States than are needed to provide an adequate medical service for the country—twice as many per unit of population as the leading countries of Europe—and the present medical schools are producing more physicians than the country needs, increasing rather than correcting the oversupply. With this oversupply, however, many rural communities are lacking proper facilities due to faulty distribution; a condition dependent largely upon economic factors, and difficult of correction.

Specialization and division of labor are inevitable; the body of knowledge is far beyond the capacity of a single individual to master. There are, however, too many specialists, and too many of those are insufficiently trained. It is suggested that some such method as a Register of Specialists be created in each state which will provide the public with information regarding those who have a suitable preparation in each field of practice.

Programs of postgraduate education by joint action of the universities and the profession are considered essential. The internship is considered as the most important and in some instances the most defective part of medical training. The medical course itself should make the student, not the courses, the credit hours, or the faculty the unit of education, for it is an axiom that all true education is self-education. Preventive medicine and instruction in positive health should receive their fair share of empha-

sis, on a par with pathology and the treatment of disease. Premedical education should be general and cultural, not too strictly preprofessional in character. The present long period of primary, secondary and college education could be materially shortened and improved through the reorganization of present courses, stimulation of better scholarship, and provision of opportunities whereby pupils with ability may proceed more rapidly.

These admirable proposals, formulated after a careful study of conditions both at home and abroad are capable of attainment, for they are largely under the control of able and thoughtful leaders. Any general reorganization of general practice, becoming increasingly essential for the good of both the public and the physician will be infinitely more difficult of attainment. Strong leadership is lacking; organization is superficial; the doctor, individualist that he is, prefers the ways of his fathers, and the American Medical Association, which should provide the leadership and the organization, prefers, like Narcissus at the pool, to admire itself as it is.

VACCINATION LAWS AND INCIDENCE OF SMALLPOX

THE table and charts* in this week's issue, showing the relation between the type of vaccination law and the incidence of smallpox in the different states, give those interested in preventing this disease some additional facts to back them up in combating the barrage of misstatements and half-truths which certain organizations are continually directing against compulsory vaccination.

Only recently a circular of misinformation in regard to the Fitchburg smallpox outbreak of a year ago has been circulated. Its wild assertions paint a lurid picture which to the uninformed, no doubt, builds up a strong case against vaccination. It actually states that six persons died as a result of vaccination, but when the authors and sponsors of the circular are requested to give the names of those alleged to have died the letter is ignored, demonstrating that the assertion cannot be backed up by facts.

It is interesting to learn that the useful facts collected in the paper referred to were gathered because the map circulated by the organization opposing vaccination suggested the idea. Thus the propagandist's own ammunition for once has been turned against him.

These figures ought to make it possible to decrease somewhat the effect of the yearly fervid plea to the legislature to "pin a gold star" on Massachusetts, since the final result of pinning on the gold star would be to give us a hundred times as much smallpox. A gold star means many a scar!

*See Page 317.

THIS WEEK'S ISSUE

CONTAINS articles by the following named authors:

DERICK, CLIFFORD L. M.D., C.M. McGill University Medical School 1918. Assistant Professor of Medicine, Harvard University Medical School. Physician, Peter Bent Brigham Hospital. Address: Peter Bent Brigham Hospital, Boston. Associated with him are

NEWTON, FRANCIS C. A.B., M.D. Harvard University Medical School 1919. F.A.C.S. Faculty Instructor in Surgery, Harvard University Medical School. Senior Associate in Surgery, Peter Bent Brigham Hospital. Address: Peter Bent Brigham Hospital, Boston. And

SCHULZ, REUBEN Z. M.A., M.D. Harvard University Medical School 1927. Instructor in Pathology, Harvard University Medical School. Address: Harvard University Medical School, Boston. And

BOWIE, MORRIS A. A.B., M.D. Harvard University Medical School 1929. Instructor in Medicine, Hospital of the University of Pennsylvania. Address: Hospital of the University of Pennsylvania, Philadelphia, Pa. And

POKORNY, NORMAN A. A.B., M.D. Johns Hopkins University School of Medicine 1929. Resident, Children's Hospital. Address: Children's Hospital, Boston. Their subject is "Spontaneous Hyperinsulinism. Report of a Case of Hyperinsulinism Cured by Surgical Intervention." Page 293.

ALDRICH, ROBERT HENRY. B.S., M.D. Johns Hopkins University School of Medicine 1929. Assistant in Surgery, Harvard University Medical School. Assistant in Surgery, Out-Patient Department, Boston City Hospital. His subject is "The Rôle of Infection in Burns." Page 299. Address: 520 Commonwealth Avenue, Boston.

AUB, JOSEPH C. A.B., M.D. Harvard University Medical School 1914. Physician-in-Chief, Huntington Memorial Hospital. Senior Associate Physician, Peter Bent Brigham Hospital. Associate Professor of Medicine, Harvard University Medical School. Address: Collis P. Huntington Memorial Hospital, 695 Huntington Avenue, Boston. Associated with him is

SMITHWICK, R. H. B.S., M.D. Harvard University Medical School 1925. F.A.C.S. Assistant in Surgery, Courses for Graduates, Har-

vard University Medical School. Assistant Surgeon to Out-Patients and Surgeon to Emergency Ward, Massachusetts General Hospital. Address: 319 Longwood Avenue, Boston. Their subject is "Lead Treatment of Cancer." Page 310.

BUNKER, JOHN W. M. A.B., A.M., Ph.D. Professor of Physiology and Biochemistry and Director of Research Laboratories in Biology and Public Health, Massachusetts Institute of Technology. Address: Massachusetts Institute of Technology, Cambridge, Mass. Associated with him are

HARRIS, ROBERT S. B.S. Research Associate, Massachusetts Institute of Technology. Address: Massachusetts Institute of Technology, Cambridge, Mass. And

EUSTIS, RICHARD S. M.D. Harvard University Medical School 1911. Instructor in Pediatrics, Harvard University Medical School. Physician to Children's Medical Department, Massachusetts General Hospital. Visiting Pediatrist at Boston Lying-in Hospital. Consulting Pediatrist at Home of Good Samaritan. Address: 319 Longwood Avenue, Boston. Their subject is "The Antirachitic Potency of the Milk of Human Mothers Fed Previously on 'Vitamin D Milk.'" Page 313.

WARREN, SHIELDS. A.B., M.D. Harvard University Medical School 1923. Instructor in Pathology, Harvard University Medical School. Pathologist, New England Deaconess Hospital, New England Baptist Hospital, Robert B. Brigham Hospital and Pondville Hospital. Director, State Tumor Diagnosis Service. His subject is "A Method for Mounting Gross Specimens." Page 315. Address: 195 Pilgrim Road, Boston.

WOODWARD, SAMUEL B. A.B., Harvard University Medical School 1878. President, Massachusetts Medical Society 1916-1919. Address: 58 Pearl Street, Worcester, Mass. Associated with him is

FEEMSTER, ROY F. A.B., Dr.P.H., M.D. Johns Hopkins University School of Medicine 1924. Assistant Director, Division of Communicable Diseases, Massachusetts Department of Public Health. Address: 546 State House, Boston. Their subject is "The Relation of Smallpox Morbidity to Vaccination Laws." Page 317.

WHEELER, WILLIAM D. A.B., M.D. Tufts College Medical School 1918. Visiting Dermatologist, The Boston Dispensary, Department of Dermatology and Syphilology and Beth Israel Hospital. His subject is "Persistently Positive Blood Reactions in Treated Syphilis." Page 319. Address: 366 Commonwealth Avenue, Boston.

CHURCHILL, EDWARD D. M.D. Harvard University Medical School 1920. John Homans Professor of Surgery, Harvard University. Chief of the West Surgical Service, Massachusetts General Hospital. His subject is "Non-Tuberculous Pulmonary Suppuration." Page 323. Address: Massachusetts General Hospital, Boston.

MASSACHUSETTS LEGISLATIVE NOTES

The following bills, of interest to the medical profession, are before the Massachusetts Legislature:

H. 106 an act relative to the qualifications of applicants for registration as qualified physicians. Public Health. Hearing February 14, Room 450, at 10:30 A.M.

H. 660 an act authorizing the Department of Public Health to distribute insulin for the treatment of diabetes and other diseases. Public Health.

S. 212 to accompany the petition of the Citizens' Committee opposing compulsory vaccinations, by John S. Codman, chairman, and another that the vaccination or inoculation of persons be made voluntary. Public Health.

H. 676 an act to promote the public health and safety by providing for the elimination of unsanitary and dangerous housing conditions, to relieve congested areas, and the construction and supervision of dwellings and for the letting of apartments at reasonable rentals. State administration.

H. 627 by Mr. Babcock of Milton, petition of Stephen S. Bean relative to premiums and interest charges on policies of insurance under the laws providing compensation for injured employees. Labor and industries.

H. 630 by Mr. Halliwell of New Bedford, petition of William E. G. Batty relative to allowance for lost time under the laws governing compensation for employees injured in industrial accidents. Labor and industries.

H. 813 by Mr. Lyons of Brockton, petition of John P. Lyons that additional persons be employed on mosquito control projects as a measure of unemployment relief. Ways and Means (Joint).

H. 749 by Mr. McCracken of Needham, petition of James W. Page and others that persons alleged to be insane be given right to examination by their own physicians in connection with hearings for discharge. Judiciary (Joint).

H. 661 by Mrs. Sweetser of Reading, petition of the Massachusetts Civic League that local public health officers and agents be examined and licensed by the Department of Public Health. Public Health.

H. 679 by Mr. Scott of West Springfield, petition of the Massachusetts County Commissioners' Association that the laws governing compensation of employees injured in industrial accidents be made to include employees of county and district hospitals. State administration.

MISCELLANY

A RADIO MESSAGE PREPARED AND SPONSORED BY THE COMMITTEE ON PUBLIC EDUCATION OF THE MASSACHUSETTS MEDICAL SOCIETY FOR THE DEPARTMENT OF PUBLIC HEALTH

CANCER*

BY CHANNING C. SIMMONS, M.D.

You have all heard a great deal about cancer recently and it is the object of the State Department of Public Health to bring the matter again to your attention. For the past 25 years through scientific investigations and the efforts of the various health departments to apply the knowledge thus gained, certain of the diseases which were accepted as uncontrollable calamities in former years have been practically stamped out. Chief among these may be mentioned typhoid fever and diphtheria. The mortality and incidence of tuberculosis have also markedly decreased. At the present time one serious disease, the deathrate from which seems to be on the increase and about which comparatively little is known, is cancer. The number of deaths from this disease is steadily increasing and as a cause of death in Massachusetts it now ranks third. Heart disease and pneumonia are the only conditions causing a greater number of deaths per hundred thousand. Tuberculosis is now in sixth place. This increase in cancer deaths has been slow but steady, and is an actual increase; the fact that more people reach adult life than formerly, and that the condition is more easily recognized with our better diagnostic facilities, having been taken into account. It is distinctly a disease of adult life and is rare before the age of forty-five.

Much work has been and is being done in the laboratories and clinics in this country and throughout the world in an attempt to discover the cause and cure of cancer. We do not know the cause, although a great deal has been learned in regard to the disease in recent years. Many theories have been advanced but none have been accepted. It is not contagious and people in close association with patients suffering from cancer are in no way liable to contract the disease. In man at least, it is probably not hereditary although a certain susceptibility is apparently transmittable. In certain animals such as mice this susceptibility to cancer is very marked in certain strains, while others appear to have an immunity. Cancer is not due to bacteria and although many have tried to prove that a given microörganism is the cause, their claims have not been substantiated by other observers. Clinically, the disease does not behave as an infectious progress. Certain men have claimed that it is due to different articles of diet, and fish, meat, raw vegetables, and even excessive water drinking has been brought forward as an etiological factor. Although we do not know the exact cause, we do know that chronic irritation is a factor of great importance in the disease. This has been proved experimentally and found to be true in practice; thus cancer of the skin is seen more commonly in people exposed to chronic irritation, such as paraffin workers, chimney sweepers, x-ray operators, etc. Poor teeth, ill-fitting dental appliances and the excessive use of tobacco have a direct relation to cancer of the mouth. Women who have had lacerations as a result of childbirth are more prone to cancer of the womb than others, and many similar examples could be given.

Pathology — As we understand the disease at present, the cancer starts as a small nest of cells in a given place and if we can recognize it at this time it can be cured. As it grows, depending on its position, it involves vital structures and seeds itself throughout the body, and if this has occurred it is impossible to cure it. This is why you are urged to seek advice at once for any unusual lump or sore. If it should prove to be a cancer, it can be destroyed before the disease spreads. In certain situations, such as the esophagus or stomach, it often causes no trouble until it is so large that nothing can be done to cure it, but in at least three-fifths of the cases the disease is visible or gives symptoms that can be recognized and leads one to seek treatment while the condition is still local and can be destroyed.

Cancer in different situations behaves in different manners; in fact there is nothing in common between cancer of the skin and cancer of the mouth. For example, in the skin it is of very slow growth and does not become disseminated for several years. On the other hand, cancer of the tongue is of very rapid growth and is often incurable in two to four months from its onset. It is unfortunate that cancer in itself in its early curable stage causes no pain or other subjective symptom. Pain, loss of weight and strength, symptoms usually associated in the mind of the ordinary person with the disease, are those of the late incurable stage.

The first indication of cancer varies as to the organ involved; thus cancer of the tongue first manifests itself as a painless ulcer; cancer of the breast as a small, hard, painless lump; cancer of the womb or rectum as bloody or other unnatural discharges. Of course, not all painless lumps or ulcers are cancer, but if they occur in an individual over forty-five years of age, it is well to consult a physician and to be reassured or receive prompt treatment.

At the present time prompt treatment of a suspicious condition or even better, the removal of any predisposing cause such as sharp teeth or dental appliances which are allowed to become rough and dirty in the mouth is the best method of combating the disease. In studying the conditions at some of the larger hospitals, it was found that the average time the patient delayed after

*Station WBZ, January 31, 1933, 4:15 P.M.

first being aware of trouble before consulting a physician was eight months. In other words, the disease had a good start before any attempt was made to eradicate it. It was found that there was a further delay of two months after consulting a physician before steps were taken to destroy the growth. It is not surprising, therefore, that the disease is often hopeless when first seen by a surgeon. In fact in nearly half of the cases admitted to the hospitals, the tumor is so far advanced that there is no hope of permanent cure.

For several years the American Society for the Control of Cancer and the State Department of Public Health have undertaken a publicity campaign in an attempt to bring before the public the early symptoms of the disease and to urge physicians to take active steps to treat any suspicious tumor, as cancer in most situations in its early stage is not an incurable disease. Statistics have shown that, if taken early, from fifty to ninety-five per cent of the cases of cancer of the organs most commonly involved can be cured. The American Society for the Control of Cancer has adopted for its slogan "In early treatment lies the hope of Cure."

As an aid to the practicing physicians in the diagnosis of early or obscure cases of cancer, the State Department of Public Health has established consultation clinics in several centers throughout the state where consultants, representing all the medical specialties, are in attendance. There are four such clinics in Boston, as well as others in Lowell, Lynn, Newton, Springfield, Worcester, Pittsfield, North Adams, Fitchburg, New Bedford, Brockton, Leominster, Gardner and Lawrence. In addition to these consultation clinics, the State has established a cancer hospital at Pondville with a capacity of 120 beds and facilities to treat the disease by the most modern methods. This hospital also maintains a consultation clinic service.

As regards treatment in its early stage, anything that will destroy the growth will cure the disease. Much experimental work on the treatment of cancer by electricity and the injections of solutions of metal, such as lead, gold, etc., has been done but at the present time only two measures are accepted, namely, surgery in some form, including the use of cautery or the so-called radio knife; and radiation, that is, either radium or x-ray. In 1927, a cancer conference was held at Lake Mohonk which was attended by many of the European cancer specialists and this was the conclusion reached as a result of the conference.

It might be well to quote the conclusions drawn up at the end of this conference, as they hold today as well as in 1927:

"Cancer in some parts of the body can be discovered in a very early stage, and if these cases are treated properly the prospect for a permanent cure is good.

"The cure of cancer depends upon discovering the growth before it has done irreparable injury to a vital part of the body and before it has spread to other parts.

"The public must be taught the earliest danger signals of cancer which can be recognized by persons without a special knowledge of the subject and induced to seek competent medical attention when any of these indications are believed to be present.

"The most reliable forms of treatment and, in fact, the only ones thus far justified by experience and observation, depend upon surgery, radium and x-ray.

"Efforts toward the control of cancer should be made in two principal directions, the promotion of research in order to increase the existing knowledge of the subject and the practical employment of the information which is at hand."

CORRESPONDENCE

ARTICLES ACCEPTED BY THE AMERICAN MEDICAL ASSOCIATION COUNCIL ON PHARMACY AND CHEMISTRY

535 North Dearborn Street, Chicago, Ill.,
January 28, 1933.

Managing Editor,
The *New England Journal of Medicine*,
Dear Doctor:

In addition to the articles enumerated in our letter of December 31, 1932, the following has been accepted:

United States Standard Products Co.
Diphtheria Toxoid—U.S.S.P.

Yours very truly,
PAUL NICHOLAS LEECH, *Secretary*,
Council on Pharmacy and Chemistry.

RECENT DEATHS

A CORRECTION

The death notice of Dr. Louis Henry Clarke which appeared in the January 26 issue, page 232, reported that Dr. Clarke died in Northampton, whereas he died at his home, 1039 Northampton Street, Holyoke.

TEMPLE — WILLIAM FRANKLIN TEMPLE, M.D., died in Boston, February 2, 1933, after a protracted illness.

Dr. Temple was born in Dorchester, December 16, 1853, the son of William Franklin and Milla (French) Temple. He graduated from Dartmouth College in 1877 and was given his M.D. degree by the Harvard University Medical School in 1881. Dr. Temple had served as physician to the Massachusetts Home for Intemperate Women and on the staffs of St. Elizabeth's Hospital, the Boston Dispensary and the Carney Hospital and was the medical officer of the Boston Protective Department for a considerable time.

He was formerly a Fellow of the Massachusetts Medical Society, having joined in 1880 and retired in 1923. He also held membership in the American Medical Association, the Harvard Medical School Alumni Association, the American Historical Association, the Medical Improvement Society, the University and Harvard Clubs.

In 1886 he married Mary Alice Ferrin of Groton who died several years ago. He is survived by three sons, Dr. William F. Temple, whose office is at 377 Beacon Street, Boston; Samuel Temple of Boston and Richard Temple of Hollywood, California.

FINKELSTEIN — HARRY FINKELSTEIN, M.D., died suddenly February 3, 1933. He was born in Russia in 1881 coming to this country early in life. He graduated from the English High School and later from the Medical School of Harvard University in 1904.

He practiced in Boston for twenty-eight years with an office at 366 Commonwealth Avenue. He joined the Massachusetts Medical Society in 1904.

Dr. Finkelstein is survived by his widow who was Miss Rose Agoos, four children, Mrs. Benjamin Goldberg of Brookline, a daughter Lazzi, two sons, Albert and George, his father Reuben Finkelstein who lives in Roxbury and two brothers, Dr. Bernard Finkelstein and Dr. Nathaniel Finkelstein, both dentists.

NOTICES

CLINICS IN MEDICINE AT PETER BENT BRIGHAM HOSPITAL

On Thursdays at 3:30 P.M. in the Amphitheatre of the Peter Bent Brigham Hospital Dr. Henry A. Christian, Hersey Professor of the Theory and Practice of Physic, Harvard, and Physician-in-Chief, Peter Bent Brigham Hospital, will give a series of clinics which members of the medical profession are cordially invited to attend. The next clinic will be given February 16.

Physicians and medical students are also invited to attend ward rounds of the Medical Staff of the Peter Bent Brigham Hospital held from 10-12 o'clock on Saturdays, as well as a clinical-pathological demonstration immediately following these rounds.

ANNOUNCEMENT

SAUL M. MARCUS, M.D., announces the opening of his office at 311 Commonwealth Avenue, Boston, Mass.

HARVARD UNIVERSITY

FREE PUBLIC LECTURES ON MEDICAL SUBJECTS

The Faculty of Medicine of Harvard University offers a course of free public lectures on medical subjects, to be given at the Medical School, Longwood Avenue, Boston, on Sunday afternoons. The lectures will begin at four o'clock and the doors will be closed at five minutes past the hour. No tickets are required.

SUNDAY AFTERNOONS AT FOUR O'CLOCK

February 12—Dr. G. H. Wright: Color Changes in the Mouth and Teeth: an Aid in Diagnosis of Systemic Disease.

February 19—Dr. W. H. Robey: Let the Head Govern the Heart.

February 26—Dr. H. R. Viets: Nerve, Nerves, and Nervousness.

March 5—Dr. E. L. Oliver: The Care of the Skin and Scalp.

March 12—Dr. Shields Warren: Cancer and Radiation Therapy.

March 19—Dr. Randall Clifford: The Tuberculosis Problem Today.

March 26—Dr. W. G. Smillie: A Discussion of the Common Cold.

RADIO HEALTH MESSAGES

FEBRUARY-MARCH, 1933

Sponsored by the Public Education Committee of the Massachusetts Medical Society and the Massachusetts Department of Public Health.

Courtesy WBZ. Tuesdays, 4:15 P.M.

February
14 Your Title to a Job
21 Why a Health Department?
28 Effects of Fatigue

March
7 Vacation Health
14 The Use and Abuse of Serums and Vaccines
21 Can We Prevent Diabetes?
28 Hobbies and Health

STATE HOUSE BROADCAST

Sponsored by the Massachusetts Department of Public Health.

Courtesy WEEI. Fridays, 12:45 P.M.

Health Subjects of Public Interest discussed by the Division Directors of the Department.

RADIO HEALTH FORUM

Queries from the public are answered under the sponsorship of the Department of Public Health.

Courtesy WEEI. Fridays, 4:50 P.M.

Questions on Health and Prevention of Disease may be sent to Radio Health Forum, State Department of Public Health, State House, Boston.

REPORTS AND NOTICES OF MEETINGS

HARVARD MEDICAL SOCIETY

The Harvard Medical Society held its stated meeting in the amphitheatre of the Peter Bent Brigham Hospital on Tuesday evening, January 10, 1933, at 8:15 P.M. Dr. Elliott C. Cutler presided.

Dr. Thompson of the Surgical Service presented the case for the evening. The patient was a 27 year old American housewife. Her past history and family history were not relevant. Four years before admission, after the delivery of her first child, she noticed a small lump, in her abdomen to the left of the umbilicus, which grew very slowly. One year previous to entry, she became pregnant for the second time, and, during the course of her pregnancy, the lump began to grow rapidly. The delivery and puerperium were comparatively uneventful, the child being born about three months previous to admission. Six to eight weeks before entry the patient was seen by one of the hospital physicians who advised a series of x-ray treatments to the very large abdominal tumor. There was some shrinkage, but following this a return to the former size. Hospitalization for radical removal was advised, and on entry the physical examination was essentially negative save for the large, non-tender, hard mass deep in the muscle layer of the abdominal wall. Laboratory findings were not contributory. The tumor was removed and pathological examination confirmed the diagnosis of a fibroblastic tumor of the desmoid type. In discussing the case, Dr. Cheever pointed out that these tumors are found sometimes in pregnant women, that they usually arise in the sheath of the rectus muscle, and that the factor of stretching of the abdominal wall may provide a stimulus for the initiation of a growth of this nature. While the tumor is apt to recur locally, unless carefully and completely removed, metastases are infrequent.

Dr. Eric Stone of Providence, Rhode Island, delivered the main address of the evening on "Medical Practices of the Aboriginal Americans." The speaker prefaced his talk with the statement that the true standing and worth of the aboriginal Americans are not generally appreciated; that the association of the Indian with what we call savagery is a myth; and that certain Indian customs, habits, and practices were superior to many of the standards of living that held among the white people during the same period.

In regard to medicine, the practices of the American Indian were at least on a par with those of the inhabitants of Europe at the time. The treatment by the Indian of diseases he understood was excellent. He has handed down to the modern "pharmacopeia" no less than 59 different drugs. The types of illness from which he suffered were very different from those afflicting us today. His diseases were mostly dietary or as a consequence of exposure. Cancer and syphilis were probably unknown among the full-blooded Indians, and there is some question as to what the situation was in regard to tuberculosis.

In practice, Indian methods may be arbitrarily divided into (1) practical and (2) supernatural. The former includes the well-established Indian practices of physiotherapy. At this point the speaker showed numerous interesting slides illustrating the methods and implements used. Sweat-baths and sweat-lodges were common, and massage, oils, and heat are frequent adjuncts to therapy. The methods of preparing and preserving medicines were next depicted on the screen. Amputation was a well-known procedure and débridement of wounds and stimulation of granulation tissue by insertion of tree bark into the wounds were both commonly done. Trephining of the skull was performed by South American Indians, but was not done in the North. The curious pictographic prescription sticks of the medicine men were next shown. These were the only medical formulae until written language (as, for example, in 1840 with the Cherokee tribe) was first used.

There were many diseases which the aboriginal American could not treat by practical means at his disposal. All the unexplained diseases were due to some kind of spirit and the treatment of such diseases involved the invocation of a stronger spirit to drive the disease-producing spirit away, or involved propitiation or cajoling of the latter, or correction of the offense. Although a symbol of a deity was often used in the ceremonials, this symbol was never confused, in the people's minds, with the deity itself.

Time and again what to us seem to be astounding cures were accomplished by supernatural or psychotherapeutic means, and the speaker believes that this influence of "mind over matter" must be a real thing, and perhaps too little recognized in our modern "white man" medicine.

At the conclusion of the talk, Miss Mary Wheelwright gave a very interesting account of some of the 18 or more, nine-day medico-religious ceremonies practiced by the Navajo Indians of New Mexico.

THE WILLIAM HARVEY SOCIETY

Dr. Morris Fishbein, Editor of the *Journal of the American Medical Association*, addressed the William Harvery Society on Thursday, January 12, on the subject of "The Art and Practice of Medical Writing." The meeting was held in the amphitheatre of the Tufts College Medical School at 1 P.M. Dr. Benjamin Spector, Associate Professor of Anatomy, introduced the speaker.

Dr. Fishbein stated that medical writing is of importance to all members of the profession. It is contrary to the long-established tradition of physicians to advertise. Good work and careful investigation, followed by publication of the results in a reputable journal, are legitimate means of bringing recognition to the physician.

If we trace the history of medicine through the ages, we find that progress has been marked by medical writing. In the papyri, the Hippocratean text and the writings of Galen are contained careful and accurate studies of disease. We can read these documents and diagnose the diseases from the lucid descriptions. They contain

the fundamentals of what are considered the criteria for good medical writing—simplicity, completeness and accuracy.

The number of medical publications today is tremendous, amounting to over 1,800, of which about 1,200 are indexed by the Cumulative Index. About 400 are published in this country.

It is of inestimable value to the student to learn how and by what standards good medical literature is evaluated, and to select from such a great array of material those publications that are significant.

In the past, papers were written largely to be read before medical societies. When reading a paper, an individual adds his personality to the text, and the listener is often favorably impressed by the material presented. Oftentimes when such a paper is read at one's leisure, it appears shoddy and poorly done. One should, in listening to papers, strive to separate the material from the personality of the speaker, and in writing papers do so with the view that they are to be read by others.

The speaker emphasized the importance of acquiring a simple style in writing. The basis of modern medical writing is the case report, and in this connection certain general principles were discussed. When attending medical school, students are frequently asked to note every finding whether negative or positive, but in writing case reports for publication only the essentials should be included. We tend to fall into the use of "rubber stamps" in medical writing. One frequently finds the word "complain" in regard to the patient's symptoms, when in reality the patient was not complaining. Such a statement as "A baby, three months of age, came into the office complaining of vomiting" is an example of the misconceptions that pattern phrases often produce. The speaker cited several examples of exuberant styles in writing as found in papers submitted to the *J. A. M. A.*

A valuable precept in writing is that "A good style lends itself easily to the rhythm of respiration." If one reads Osler's medicine one finds that every sentence has rhythm. The writings of Pasteur, Virchow, Beaumont and others are characterized by simplicity, clarity and a pleasant style.

Medical jargon tends to creep into the literature from the clinic. We often refer to a patient with heart disease as a "cardiac"; we refer in the clinic to patients with venereal infections as "luetics," and patients with gonorrhea as subject to Neisserian disorder in order to spare their feelings. Although this practice may be commendable, it need not be carried into medical writing. Abbreviations such as L.L.L.N.R., meaning left lower lobe no râles, may be only local in their usage, and may cause great consternation in the minds of those in foreign countries who read the publication.

In writing most papers one should give his contribution, and not attempt to summarize everything that has been done before. If a recent résumé of the literature has been made, it may be referred to, and then the new material presented. In the numbering of reported cases one should begin with number one and not use the hospital number, which lends the idea of exaggeration and advertising.

Titles of articles are often amusing, and, as an example of this, several titles were quoted by the speaker—"Idle Thoughts on Medicine," "The Macedonian Call," "Foci of Infection Above the Collar Bone." The title should not aim at cleverness, but rather state in a few words just what the paper attempts to present. If a title is misleading and poorly composed, one is tempted to suspect the text of falling into a similar category.

We do not "operate a patient," but "operate on a patient". We do not "cystoscope a patient," but "perform a cystoscopy on a patient". A New England practitioner once wrote that he had "obstetricated a patient". Another common error is to state that a patient had no temperaure instead of saying that he had no fever.

When the manuscript is complete, one should finish with a summary and conclusions, or perhaps these may be the same in some instances. In the summary, one should briefly restate the material that has been presented, and the conclusion should note the essential contribution of the text to medical knowledge.

GREATER BOSTON MEDICAL SOCIETY

The Greater Boston Medical Society held its stated meeting in the Beth Israel Hospital Auditorium on Tuesday evening, January 3, 1933. Dr. William Dameshek presided. Dr. Elliott C. Cutler, Moseley Professor of Surgery at the Harvard Medical School, addressed the Society on the subject of "Postoperative Pulmonary Abscess, Experimental and Clinical Studies."

Dr. Cutler said that he wished to discuss postoperative pulmonary abscess in particular, because it is an example of a condition that, despite the control of hemorrhage and sepsis, constitutes grave problems for the surgeon. He spoke from an historical viewpoint, and told of his own laboratory experiments.

All surgical procedures before the discovery of ether anesthesia were necessarily confined to emergency conditions. With the use of ether, in addition to deaths from sepsis, there were many so-called "ether pneumonias". After the use of cocaine, as a local anesthetic in 1879, and the introduction of spinal anesthesia in 1884, Mikulicz early claimed that many postoperative pulmonary complications followed the use of cocaine. This was the first suggestion that the name "ether pneumonia" would have to be discarded. In 1908 Ranzi published an important paper on the "Em-

bolic Origin of Postoperative Pulmonary Complications."

In 1915-1916 Dr. Cutler's study with Dr. John J. Morton at the Massachusetts General Hospital, of what appeared to be a striking number of postoperative pulmonary complications, suggested that while the bad anesthesia of the day was to some degree responsible, embolism from the operation field was perhaps an equal if not greater factor in the production of postoperative pulmonary complications. Other causes might have been atelectasis, aspiration, decreased resistance of the host, etc. Anesthetists had been blamed for the pulmonary complications, but with other than inhalation anesthesia, the only thing common to every surgical procedure, it became increasingly clear that the surgeon had much to do with lung complications.

Furthermore, the more rough and careless the operator, the more common seemed to be postoperative pulmonary complications. Exquisiteness of technique is a prime requisite for elimination of pulmonary complications. Thrombi are formed in the operative field due to stasis, injured intima, presence of bacteria, and tissue juices. All vessels must be treated carefully; just as one important example, in tying off a tributary vein which goes into a larger one, it is probably quite important to tie it off at the junction, and not leave a reservoir of non-circulating blood in an injured vein by tying it above the point where the two join.

The experiments of Holscher, Kelly, Myerson, Lemon and others have stressed the etiologic importance of the inhalation of buccal contents in lung abscess. Dr. Cutler admits that it is easily demonstrated that substances such as methylene blue placed in the buccal cavity enter the lungs during most operations, but adds that this does not necessarily mean that they cause damage, since the endobronchial tree is very resistant.

It has long been known that movement of the operative field is of direct significance in postoperative pulmonary complications. The incidence of pulmonary complications after operation is one and one-half to two per cent; in wounds near the diaphragm, the incidence is higher. In orthopedic and neurological surgery it is lower, for in these fields there is almost complete immobilization of the parts.

In a review of 1,900 cases, Dr. Cutler found that 30 per cent of lung abscesses were postoperative, that 15 per cent followed tonsillectomy. The great number found following tonsillectomy is due to the fact that 25 per cent of operations in a septic field is for removal of the tonsils. Also, abscesses do occur when the cough reflex is still present, which at least suggests that inhalation, even in tonsillectomies, may be overemphasized.

Believing from his studies that septic embolism from the operation field was the important factor in postoperative lung abscess, Dr. Cutler was led to experimental studies on abscess production in dogs. In this he kept trying to simulate nature, because, although many workers had previously inserted innumerable kinds of bacteria into the bronchial trees of animals, with and without foreign matter, many of these experiments lacked value because of difference from the human situation.

In 1925 a long series of experiments began. First, bacteria, and other foreign substances injected into the bronchial tree via the mouth, produced no lung abscesses in dogs. Experiments with septic emboli followed. First gelatin capsules containing blood, bacteria, and a small piece of lead filing to facilitate following the embolus with the x-ray, were placed in the jugular vein. Emboli made by infecting a segment of femoral vein with pathogenic organisms were placed in the jugular vein. Abscesses were producible in 90 per cent of the experiments, with healing in about two and one-half weeks as a rule. Incidentally, most of the lesions occurred in the left lower lobe, due to the nature of the arteries supplying the lungs.

In an attempt to simulate nature more exactly, infected clots were prepared in capillary tubes, and slipped into the jugular veins of dogs. It was indicated that the femoral vein segments of the previous operations had been barriers to the spread of infection once established in the lung, and that the new method was apt to produce a widespread pneumonitis. However, the reactions in the dogs' lungs varied from a widespread pneumonitis, with death in a few days, to tiny infected foci which rapidly healed. The factors influencing the condition were the immunity of the host and the virulence of the organisms.

Next abscesses were created in the neck in the region of the jugular vein, and operation and traumatization of tissues and vessels were accomplished. These experiments failed to create a typical lung abscess, though some results followed when veins were traumatized and when fibrinogen and bacteria were injected. An infected clot was produced in the jugular vein of a dog with a neck abscess and then half was allowed to enter the circulation and half was placed in the jugular vein of another animal. In the former animal some immunity had been established and there was not much effect, but in the newly injected animal a widespread pneumonitis was produced, indicating the existence of the relative immunity of the host versus virulence of the organism.

Hitherto no chronic pulmonary abscesses had been produced in dogs. All the abscesses healed soon or produced widespread infection and death. Archibald and others had pointed out that this showed differences between human beings and dogs, due to position of the body, and that human beings cough more than do dogs. Dr. Cutler found that dogs with small abscesses, made to cough by being put in chlorine chambers, developed larger and longer-lasting abscesses.

The bacteriology of chronic abscesses was next investigated and, as is well known, anerobic bacteria and spirochetes were usually isolated. Dr. Cutler became convinced that a small abscess due to a septic embolus might form and subsequently break into the bronchial tree and then become secondarily infected with anerobes and spirochetes from the mouth. With this pathological sequence in mind the study was carried out artificially in dogs, and chronic abscesses lasting 100 days were produced.

He believes that the situation, outlined above, obtains in human beings. Most human postoperative lung abscesses occur after invasion of the lung by septic emboli, breaking into the bronchial tree, and secondary invasion from the mouth above. The speaker is convinced that 50 per cent of pulmonary "abscesses" remain in the first stage and never become chronic. Septic embolism is not the only factor, although it is perhaps the greatest one. In varicosities of the leg, there is not much opportunity of separation of septic emboli, owing to the presence of so many valves. The pelvis, however, with no venous valves, is the notoriously fertile field for the sowing of septic emboli which will invade the lungs.

Slides were shown demonstrating his technique of introducing septic emboli into the jugular veins of dogs and methods of following the resulting lesions by x-ray. He showed x-ray plates of three human cases, pointing out that where the mouth is clean and free from spirochetes, abscesses, even though they develop and show typical x-ray films, usually disappear quickly.

In conclusion, Dr. Cutler emphasized the fact that we must always try to reproduce nature as closely as possible in experiments, and that this work has been of aid to prognosis; if the abscess is seen early, and the mouth kept clean and spirochetes eliminated by a short course of arsenic, the prognosis is good.

Dr. Harry Linenthal opened the discussion. In 41 cases of abscess of the lung seen at the Beth Israel Hospital, 23 were postoperative. Of these 17 followed tonsillectomy, all in adults; four followed extraction of teeth, and two followed cholecystectomy. Although he came prepared to stress the importance of aspiration in the etiology of postoperative pulmonary abscess, the speaker admitted that he was quite convinced of the septic embolus factor after Dr. Cutler's talk. Dr. Linenthal believes that nose and throat men ought to insist on more prophylaxis and cleanliness preoperatively and always "clean up" the prospective field as much as possible. He also thinks that more coöperation between medical men, surgeons, and bronchoscopists is necessary, especially in the establishment of clear criteria as to when and how to treat a once established lung abscess.

Dr. Louis M. Freedman pointed out the function of bronchoscopy as an adjuvant in the diagnosis and treatment of pulmonary abscess and said that the use of this instrument is always to be considered in these cases. Dr. Rapoport emphasized the importance of inhalation, especially after vomiting of recently ingested food, in the production of abscess, and added that he believes abscess to be more common after general inhalation anesthesia than after local or spinal.

In closing the discussion, Dr. Cutler warned of the treachery of figures, pointed out that the figures, of tonsillectomy or of any other operation, are useless unless *all* the cases are followed by actually seeing the patient from time to time. He cannot fully explain the infrequency of postoperative pulmonary abscesses in children, as brought out in one question, but believes it may be due to cleaner mouths in the young.

The speaker stated that the ideal therapy is (1) prophylaxis in so far as this is possible. If the mouth is "dirty" and contains spirochetes, neoarsphenamine should be used preoperatively and postoperatively. Very careful handling of tissues during the operation is highly essential. (2) Early recognition and treatment of abscesses, potentially chronic, are advisable. (3) Once started, an abscess is a medical lesion and is to be treated by postural drainage and other medical means. Perhaps bronchoscopy and actual washing out of the cavity with arsenic solution may be a good procedure. (4) The surgeon should always wait in cases of pulmonary abscess and be in no hurry to operate. In chronic, long-standing, well walled-off, peripheral abscesses, surgery is indicated. (5) Hilar abscesses are a very serious problem. If bronchoscopy and postural drainage are of no value, collapse of the lung should be tried to see what it will accomplish. Then thoracoplasty may be done. Lobectomy is not to be considered in the treatment of pulmonary abscess—it is a fatal procedure in this condition.

THE BOSTON TUBERCULOSIS ASSOCIATION

Annual Meeting

At the Twenty-Ninth Annual Meeting of the Boston Tuberculosis Association of 554 Columbus Avenue, Boston, January 27, held at 4 P.M., at the Sheltered Workshop of the Association, 35 Tyler Street, Boston, Dr. John B. Hawes, 2d, President of the Association, made the following report:

Prendergast Preventorium

A total of 166 children were cared for and forty-nine new families represented. These children of thirteen different nationalities came from every district in Boston. In February, 1932, owing to lack of funds, ten children were sent home. Thanks to the generosity of two friends each giving $1,000 and some contributions of smaller amounts, these children were reinstated and forty beds maintained during the year. The School Department of Boston provides teachers for the open-air school. Classes in manual training and cooking are being

maintained. No serious illness during the year is reported. Under the direction of a teacher the children develop gardens. These children took first prize at the Horticultural exhibit for the "gardens of children contest." Six hundred and forty-eight home visits were made by follow-up nurses.

WARD 9

The death rate in this area is now said to be second in the city. The health education work is aggressive in this Ward. Classes in Home Hygiene and Care of the Sick are given during nine months of the year. The office of the Boston Tuberculosis Association was used for lecture service and demonstrations by an interior decorator from the Better Homes Association and by the Assistant Secretary of the Horticultural Society. Members of the classes and clubs in Ward 9 were urged to make use of clinics for examination and advice. Groups from Ward 9 were taken to the Prendergast Preventorium and to the Sheltered Workshop and these visits made a great impression.

SOUTH END MEDICAL CLUB

This club is now six years old and has held nine luncheon meetings during the year. It is self-supporting. The average attendance was twenty-nine. The lectures were given by the most prominent doctors in Boston.

TRUDEAU SOCIETY

There were two meetings during the year, one at the Beth Israel Hospital and one at the Middlesex County Sanatorium.

CHINESE WORK

The death rate from tuberculosis is said to be highest in Ward 3. The Chinese people have not been using the city clinics to any great extent. Therefore a clinic for them was started in April, 1932. One hundred and twenty-four Chinese have been examined and five found to have tuberculosis. This is the first time in the history of Boston's Chinatown that a place has been set aside especially for medical work among the Chinese. They are conservative and new ideas are not adopted readily. It is therefore a remarkable achievement to have 124 Chinese people come to this clinic to be examined.

PUBLICITY

The editors of the different Boston newspapers have been most generous and considerate in their help, and a far greater drop in the Christmas Seal Sale would have been felt had not the papers shown us such coöperation. The value of this publicity has been proved in donations of money and clothing to help us carry on the work. The publicity has increased more than one-third over that of last year.

BUREAU OF INFORMATION

The total number of people who came to the office for information was 850. Telephone calls for the same service totaled 450. A sum was donated by a member of the Association to assist those persons who were in great need. This Bureau, giving advice as it does on clinic service, sanatorium care and referring patients to relief agencies, is of the greatest importance. It is a most human service which this Association renders to Boston people.

LECTURE SERVICE

Five hundred and fifty lectures on tuberculosis were given before schools of nursing, men's and women's clubs, parent-teacher associations and to lay groups, with and without motion picture films and slides given by doctors and members of the staff.

REPORT OF THE SHELTERED WORKSHOP

Dr. Hawes reported that at the Sheltered Workshop of the Association 50 patients have been employed. Seventeen patients were discharged, 10 of them well enough to resume their old work. Clinics were held twice a month at the shop. One hundred and forty-four examinations were made. All patients were supervised in their homes and adequate subsidy procured for those who were physically unable to support themselves. Vacations in the country were provided for those who especially needed them. There is now a waiting list of thirteen.

Dr. Hawes thanked the individuals, churches, clubs, and organizations which helped the Association during the year and enabled it to carry on its work without a deficit.

In spite of the fact that the returns from the sale of Christmas Seals has dropped 35 per cent in the past two years, the work of this Association must go on.

The following officers were elected:

President, Dr. John B. Hawes, 2d; Vice President, Dr. James J. Minot; Treasurer, George S. Mumford; Clerk, Mrs. Reginald Heberwhite.

The Executive Committee includes, in addition to the officers named above, Adolph Ehrlich, Dr. Cleaveland Floyd, Dr. Donald S. King, Henry C. McKenna, Everett Morss, Jr., Dr. Edward O. Otis, Dr. Joseph H. Pratt, Miss Julia C. Prendergast, Alexander Wheeler, Dr. Charles F. Wilinsky and Dr. Nathaniel K. Wood.

BICKUR CHOLIM HOSPITAL STAFF

The regular monthly meeting of the Bickur Cholim Hospital staff was on January 30, 1933, in the nurses' home of the Hospital.

Dr. D. Davis, who is assistant in Medicine at Harvard Medical School, and Research Fellow at Beth Israel Hospital, demonstrated with films a long line of cardiographs, normal and pathologic, demonstrating the significance of the different waves as means of establishing the correct diagnosis in diseases of the heart. A long discussion and many pertinent questions were satisfactorily answered. Luncheon followed the meeting.

M. J. Konikow, M.D., *Recorder.*

MASSACHUSETTS ASSOCIATION OF BOARDS OF HEALTH

At a meeting held January 26 the following officers were elected: Joseph R. Glennon, president; Dr. George H. Bigelow, first vice-president; Jacob R. Sackett, second vice-president; George T. Lennon, secretary-treasurer.

NEW ENGLAND OPHTHALMOLOGICAL SOCIETY

At the Annual Meeting of the New England Ophthalmological Society on January 17, 1933, the following members were elected to office:

Dr. Hugo B. Riemer, President.
Dr. Samuel J. McDonald, Vice-President.
Dr. Benjamin Sachs, Secretary-Treasurer.
Dr. James J. Regan, Reporter.

BENJAMIN SACHS, M.D., *Secretary.*

THE NEW ENGLAND PHYSICAL THERAPY SOCIETY

The regular January meeting of the New England Physical Therapy Society was held in the Music Room of the Boston Square and Compass Club on January 18, 1933. A Round Table dinner preceded the meeting.

Dr. William G. Curtis was program chairman for the meeting, which began with a presentation of cases. Dr. Baxter raised a question regarding the treatment of a case of diabetes insipidus. Dr. Carr described such a case which responded favorably to treatment by autocondensation.

The principal speaker of the evening was Dr. Francis P. McCarthy, dermatologist at the Boston City Hospital and pathologist at St. Elizabeth's Hospital and Quincy City Hospital, who spoke on "The Use of Physical Therapy in Dermatological Conditions." He discussed the histopathology of various skin lesions and indicated the value of biopsy specimens in order to make an absolute diagnosis in many cases. Dr. McCarthy spoke on his own experience in the use of the high frequency current, the ultraviolet light, and the use of carbon dioxide snow, in the treatment of various skin conditions. He considered the high frequency current the most valuable of the modalities used in dermatology for the destruction of various lesions of the skin and mucous membrane. The dosage of the high frequency current can be readily controlled, but requires a considerable degree of experience which can be obtained only by its use in a large dermatological clinic. It is employed in the removal of various types of warts, keratotic lesions, naevi, and malignant neoplasms. He gave an interesting description of the many kinds of verrucae and differentiated the various cases of keratoses, describing particularly the senile and seborrheic type.

The various types of leukoplakia were described, according to their grade, both from a clinical and pathological standpoint, and the use of the high frequency current in these lesions was recommended. A luetic type of leukoplakia, involving the tongue, which arises from an atrophic glossitis was described and the futility of local treatment was shown by the prompt return on removal. The growing prevalence of leukoplakia, due to the products of combustion as a result of the increasing use of tobacco has been noted in recent years.

Ultraviolet therapy has proved of value in at least five diseases: acne vulgaris, pityriasis rosea, psoriasis, chronic ulcers, and telangiectasia. It has proved useful in another group of from 20 to 30 skin conditions. The indiscriminate use, however, of both natural sunlight and the ultraviolet lamp is attended with some danger. Its tendency to activate a latent malignancy of the skin is well known especially in old people. The danger, especially in blondes, of causing lupus erythematosus was mentioned.

Dr. McCarthy followed his talk by a series of lantern slides, showing various types of verrucae, naevi, keratoses, leukoplakia, and malignancies of the skin and mucous membranes.

A discussion followed which was opened by Dr. George E. Percy, who reported several favorable cases treated by the desiccating current or ultraviolet lamp. He stressed the importance of small dosage extended over a long period of time. Dr. C. L. Payzant discussed a case of pemphigus which was cured by a series of twelve colonic irrigations. He suggested that pemphigus may be due to autointoxication. If pemphigus is diagnosed early and colonic irrigation immediately instituted, the case may be cured. He discussed a case of recurrent rodent ulcer which was aggravated by radium and recurred after fulguration and local excision by electrosurgery. A more radical excision has finally apparently cured the patient.

Dr. McCarthy closed the discussion and defined dermatology as something more than the mere use of mechanical modalities; that a diagnosis must be made of the condition, and the proper use of topical applications and internal remedies prescribed; and that the use of the various electrical devices, namely, x-ray, ultraviolet, and high frequency current, should be restricted to selected cases.

NORFOLK DISTRICT MEDICAL SOCIETY

The next meeting of the Norfolk District Medical Society will be held February 28 in the Milton Club, Central Avenue, Milton, Mass., at 8:30 P.M. The subject will be "The Diagnosis and Treatment of Anemia," by Dr. William P. Murphy.

FRANK S. CRUICKSHANK, M.D., *Secretary.*

SOUTH END MEDICAL CLUB

The next regular meeting of the South End Medical Club will be held at the office of the Boston Tuberculosis Association, 554 Columbus Avenue, Boston, on Tuesday, February 21, 1933, at 12 noon.

The speaker will be Dr. William E. Brown, Junior Visiting Surgeon, Carney Hospital, and his subject, "Contusions, Strains and Sprains in Automobile and Industrial Accidents." The usual luncheon will be served at 1 o'clock. All physicians are cordially invited to attend. Come and bring guests who are in the medical profession.

MASSACHUSETTS GENERAL HOSPITAL

A Clinical Meeting of the Staff of the Massachusetts General Hospital will be held in the Moseley Memorial Building, on Thursday, February 16, 1933, at 8:15 P.M.

PROGRAM

1. Malignant Goiter—Dr. Allen Graham, Cleveland, Ohio.
2. Degrees of Malignancy in Thyroid Cancer—Dr. Shields Warren.
3. Some Observations on Adenomas of the Thyroid—Dr. Jacob Lerman.

Physicians, Medical Students, Nurses and Social Workers are cordially invited.

COMMITTEE ON HOSPITAL MEETINGS.

NEISSERIAN MEDICAL SOCIETY OF MASSACHUSETTS

The Neisserian Medical Society of Massachusetts will hold its seventh meeting on Friday, February 17, 1933, at the Beth Israel Hospital, Boston, at 8 P.M.

The meeting will be devoted to the discussion of plans to be carried out by the Society, the purpose of which is to bring about an improvement in the control of gonorrhea in the community.

The medical profession is cordially invited to attend.

OSCAR F. COX, JR., M.D., *Secretary.*

ANNUAL TUFTS COLLEGE MEDICAL SCHOOL DINNER

The annual dinner of Tufts College Medical School, for students, faculty and alumni, will be held Monday evening, February 20, 1933, at 6:30 o'clock in the main dining room of the University Club, Boston. Preceding the dinner at 5:30 o'clock, the annual meeting of the alumni association will be held. President John A. Cousens, Dean A. Warren Stearns and several alumni and department heads will speak. Dr. Louis E. Phaneuf, Professor of Gynecology, will preside. Reservations for the dinner may be had by addressing the Tufts College Medical School Dinner Committee, 416 Huntington Avenue, Boston.

NEW ENGLAND PHYSICAL THERAPY SOCIETY

The regular meeting of the New England Physical Therapy Society will be held in the Music Room of the Boston Square and Compass Club, 448 Beacon Street, Boston, at 8 P.M., on February 15.

The program for this meeting is in charge of Dr. Edward H. Trowbridge.

PROGRAM

Diathermy in Urethral Growths and Erosions of the Cervix (with motion pictures), Edward H. Trowbridge, M.D., Worcester, Massachusetts.

Discussion to be opened by DeWitt G. Wilcox, M.D.

As a pioneer in the use of electrosurgery and electrical currents in the treatment of disease, Dr. Trowbridge has contributed much to the development of physical medicine. His paper will be of great value and the meeting worthy of a large attendance.

A council meeting is scheduled for five o'clock. As there are several important matters to be considered, councilors are urged to arrive promptly.

There will be the usual Round Table Dinner at six-thirty.

All members of the medical profession are invited to attend and to participate in the discussion.

ARTHUR H. RING, M.D., *Secretary.*

AMERICAN ACADEMY OF PHYSICAL THERAPY

SPECIAL MEETING

A special meeting of the American Academy of Physical Therapy will be held at 2 P.M. on Saturday, February 18, at the Hotel Statler in Boston.

The purpose of this meeting is to vote upon proposed changes in the By-Laws and to transact other important business.

All Fellows of the Academy are urged to be present, if possible.

ARTHUR H. RING, M.D., *Secretary-Treasurer.*

WATERTOWN PHYSICIANS' SOCIETY

The regular monthly meeting of the Watertown Physicians' Society will be held Tuesday, February 14, 8:30 P.M., at 128 Mt. Auburn Street.

The speaker of the evening will be Dr. Charles N. Gettes. Subject: "Ear Pathology."

Physicians interested are cordially invited to attend.

EUGENE F. GORMAN, M.D., *Secretary.*

NEW ENGLAND HOSPITAL FOR WOMEN AND CHILDREN

The regular clinical conference of the New England Hospital for Women and Children will be held at the hospital, Dimock Street, Roxbury, on Thursday, February 16, at 8 P.M. Dr. Ettinger of Germany, who is doing research work at the Boston Dispensary, will speak on "The Relief Method of X-ray in Gastro-Intestinal Disease," and will illustrate her talk with slides.

ALICE H. BIGELOW, M.D., *Secretary.*

THE NEW ENGLAND ROENTGEN RAY SOCIETY

The February Meeting will be held at the Massachusetts General Hospital in the lower amphi-

theatre of the Out-Patient Department, Friday, February 17, 1933, at 8:15 P.M.

SCIENTIFIC SESSION

Dr. George W. Holmes will present the following program, a series of short papers (ten minutes each), by members of the staff of the X-ray Department of the Massachusetts General Hospital:

Incidental Findings in Routine Heart Examinations. J. Maurice Robinson, M.D.

Calcification of Traumatic Origin Within the Skull. John Meachem, M.D.

Some Unusual Types of Lobar Pneumonia. Robert G. Vance, M.D.

The Distribution of Bone Metastases in Carcinoma of the Breast. Richard Dresser, M.D.

Some Early Results of "Spray Treatment." Stevens S. Sanderson, M.D.

Spasm of the Pyloric Antrum in Gastric Ulcers. James R. Lingley, M.D.

Two Cases showing Deformity of the Duodenal Bulb. Ernest G. Smith, M.D.

Spastic Abnormalities of the Gastro-Intestinal Tract Due to Lead Poisoning. William C. Martin, M.D.

Abnormal Gas and Soft Tissue Shadows in the Abdomen and Pelvis. Aubrey O. Hampton, M.D.

T. R. HEALY, M.D., *Secretary.*

370 Marlborough Street, Boston.

SOCIETY MEETINGS

CONGRESSES AND CONFERENCES

February 10—William Harvey Society. See notice on page 290, issue of February 2.

February 12 - March 26 — Harvard University. Free Public Lectures on Medical Subjects. See page 339.

February 14—Watertown Physicians' Society. See page 346.

February 14—Harvard Medical Society. See page 289, issue of February 2.

February 15—New England Physical Therapy Society. See page 346.

February 16—Clinics in Medicine at Peter Bent Brigham Hospital. See page 339.

February 16 — Massachusetts General Hospital. See page 346.

February 16—New England Hospital for Women and Children. See page 346.

February 17—New England Roentgen Ray Society. See page 346.

February 17—Neisserian Medical Society of Massachusetts. See page 346.

February 17 and 18—The New England Hospital Association. See notice on page 911, issue of November 17.

February 18—American Academy of Physical Therapy. See page 346.

February 20—The Annual Tufts Medical School Alumni Dinner. See page 346.

February 21—South End Medical Club. See page 345.

April 1 and June 13—The American Board of Obstetrics and Gynecology. See page 233, issue of January 26.

April 5—New England Heart Association. See notice on page 1011, issue of December 1.

DISTRICT MEDICAL SOCIETIES

ESSEX NORTH DISTRICT MEDICAL SOCIETY

May 3—Wednesday, Annual Meeting. Program to be announced.

ESSEX SOUTH DISTRICT MEDICAL SOCIETY

Tentative Program

Wednesday, March 8, 1933—Lynn Hospital. Clinic 5 P. M. Dinner 7 P. M. Speaker: Dr. Soma Weiss, Boston City Hospital. Subject: Arterial Hypertension.

Wednesday, April 5, 1933—Essex Sanatorium, Middleton. Clinic 5 P.M. Dinner 7 P.M. Speaker: Dr. Wilson G. Smillie, Professor of Public Health Administration, School of Public Health, Harvard University. Subject: Common Colds.

Thursday, May 4, 1933—Censors' Meeting, Salem Hospital. 3:30 P. M.

Tuesday, May 9, 1933—Annual Meeting. Salem Country Club, Forest Street, Peabody. Golf in afternoon; greens fee $2.00. Dinner at 7 P. M. Speaker: Dr. George Burgess Magrath. Subject to be announced later. Ladies invited.

R. E. STONE, M.D., Secretary.

FRANKLIN DISTRICT MEDICAL SOCIETY

Meetings will be held at the Weldon in Greenfield on the second Tuesday of March and May.

CHARLES MOLINE, Secretary.

MIDDLESEX EAST DISTRICT MEDICAL SOCIETY

The program of the Middlesex East District Medical Society for the coming year is as follows:

March 15 at Melrose.

May, at the Meadowbrook Club, Reading.

ALLAN R. CUNNINGHAM, Secretary.

NORFOLK DISTRICT MEDICAL SOCIETY

February 28, 1933—See page 345.

March 28, 1933—Time, place and subject to be announced. Speaker will be Dr. J. P. O'Hare.

May—Annual Meeting. Time, place and subject to be announced.

FRANK S. CRUICKSHANK, M.D., Secretary.
1695 Beacon Street, Brookline.

SUFFOLK DISTRICT MEDICAL SOCIETY

Wednesdays at 8:15 P. M.

March 29, 1933—Clinical Program, Massachusetts General Hospital.

April 26, 1933—Annual Meeting. Election of Officers. Scientific Program—Titles and speakers to be announced later.

The Medical Profession is cordially invited to attend these meetings.

WILLIAM H. ROBEY, M.D., President,
LELAND S. McKITTRICK, M.D., Secretary.

WORCESTER DISTRICT MEDICAL SOCIETY

Wednesday, March 8—Dinner and scientific program at the Memorial Hospital, Worcester. Program to be announced later.

Wednesday, April 12—Dinner and scientific meeting at the Rutland State Hospital. Scientific program to be announced later.

Wednesday, May 10—The Annual Meeting. Place and program to be announced later.

ERWIN C. MILLER, Secretary.

BOOK REVIEWS

Preventive Medicine. By MARK F. BOYD, M.D., M.S., C.P.H. Member of the regular field staff, International Health Division of the Rockefeller Foundation, formerly Professor of Bacteriology and Preventive Medicine in the Medical Department of the University of Texas. Fourth Edition. Published by W. B. Saunders Company, Philadelphia and London. 1932. Price $4.50.

This is the fourth edition of a familiar book. Its scope and character are indicated by the author in his "Foreword" as follows:

"The following pages represent an endeavor to briefly present the salient features of modern preventive medicine. In a desire to cover the entire field, a great condensation has been effected, which in many instances perhaps results in an inadequate consideration of important subjects. It is believed this represents the minimum knowledge of the subject which a student of medicine or a practitioner of medicine should be expected to possess." . . . "No originality is claimed for the material here

presented. All available sources of information have been freely utilized."

The author has, however, not failed to give credit to his sources of information and a most valuable feature of the book is the list of "References" to be found at the end of each chapter.

The author sets a high standard of knowledge for the student and medical practitioner in expecting them to remember everything considered in this book, even until they might discover, as expressed by an old American humorist, that they "knew too many things that are not so", for "important advances in knowledge in fields included in the scope of preventive medicine", which, as the author says, have necessitated this extensive revision of his book, will, it is to be hoped, still continue and call for further revisions.

The author certainly has succeeded in presenting with considerable detail a wide variety of subjects within 500 pages, and, notwithstanding the credit which the author gives to others, he has not merely quoted from his "references", but has digested what they have to say, and has presented in his own way the material which they offer.

The author divides the book into the following "Sections", each Section being subdivided into several chapters; Diseases, due to invading microörganisms (23 chapters), Occupational Diseases, The Puerperal State, Heredity and Disease, Special Aspects of Hygiene and Sanitation (4 chapters), Demography, Public Health Administration.

Whatever the author's practical experience may have been, his book is essentially a textbook written and arranged by a teacher. It will be noted that in some instances the author is more dogmatic and in others less specific than a clinician or an experienced local public health executive would be. The following illustrations will suffice.

The public health importance of influenza calls for no apology for its selection for this purpose. In discussing influenza and its control the author recognizes that mortality in influenza is due to secondary infections, but does not say specifically, as an experienced medical practitioner would have said, that the way to reduce mortality from influenza is to isolate the sick and sterilize their eating and drinking utensils for the patients' own sake and stop putting them in rooms and hospital wards with cases of pneumonia or with streptococcus carriers. In connection with influenza also it may be noted that the "period of communicability" is stated to be "indefinite and uncertain, probably for at least seven days from the onset". No mention is made of the clinical and experimental evidence that influenza, like most if not all acute communicable diseases, is contagious in the prodromal stage before diagnostic symptoms appear, and that the infective agent of influenza ceases to be infective to other persons soon after the fever becomes pronounced. There are those who would also take exception to the author's statement that catarrhal symptoms are an accompaniment of an uncomplicated influenza infection.

The author stresses at considerable length the importance of the reporting of communicable diseases by physicians. He states that "the health officer should endeavor to discover the source of infection of every case of reportable disease recognized, simultaneously with the inauguration of appropriate control measures". Nowhere in connection with control measures, however, does he mention, as a practical health officer would, our dependence upon the thermometer to detect infected contacts in a prodromal infective stage of the disease before diagnosis can be made if we are to hope to prevent the person from causing all the trouble that he would be likely to cause. No reference is made to the part which natural immunity plays in the supposed success of ordinary quarantine measures.

The following are quotations from the author's introduction:

"Preventive medicine may be defined as that branch of applied biology which seeks to reduce or eradicate disease by removing or altering the responsible etiologic factors.

"Since preventive medicine requires a complete knowledge of the etiology of disease for its application, it is apparent that deficiencies of etiologic knowledge must necessarily limit the scope of successful work." There are, however, five groups of diseases whose etiology is sufficiently well known to warrant their classification as preventable. "The group consists of the following:

"(I) Diseases produced as the result of the invasion of the body by microörganisms.
"(II) Diseases the result of a faulty or deficient diet.
"(III) Diseases the result of unhygienic or insanitary conditions of employment.
"(IV) Diseases arising as the result of the puerperal state.
"(V) Diseases transmitted from parent to offspring."

The following is taken from the concluding paragraph of the author's "Introduction":

"The results which can accompany the application of the principles of preventive medicine received their first great popular demonstration by Gorgas when he eradicated yellow fever and malaria from Havana and the Canal Zone."

Were General Gorgas alive and this statement brought to his attention he would not let it pass without calling attention to the fact that Surgeon Joseph H. White of the United States Public Health Service first demonstrated in New Orleans in 1905, and received credit therefor, that a yellow fever epidemic could be stopped in warm weather in a susceptible population by the application of Major Walter Reed's demonstration of its mosquito transmission and that the files of the United States Public Health Reports show cases of yellow fever to have been reported in Havana after that date; that malaria has never yet ceased to be reported either

in Havana or in the Canal Zone; that during the construction of the Canal the antimalaria campaign did not vary in any important particular from the plan formulated for the purpose at the beginning of construction work by an already world-wide recognized authority on malaria prevention, Surgeon Henry R. Carter of the United States Public Health Service; that the reason why the constantly occurring cases of malaria did not show the serious effects of the disease as in the "French days" was due to progress in clinical medicine—to more effective treatment resulting from deductions from the discoveries of Manson and Ross regarding the life cycle of the malarial parasites.

While one may find reasons to criticize the author's presentation of his subject in certain particulars, the fact that the book has passed into its fourth edition sufficiently attests its popularity and usefulness.

The Mongol in Our Midst. By F. G. Crookshank. Third Edition. Kegan, Paul, Trench, Trubner & Company. London, 1931. Pp. 539. Price 21s. net.

This book is not an easy one to review and perhaps cannot be adequately reviewed by anyone except the trained anthropologist. While it has much of interest and importance for general medicine, and particularly for psychiatry, much of the material is unfamiliar to the average medical man.

A number of ideas are put forth and reasons given for them which are at variance with the established opinions of anthropology. Under such circumstances, the reviewer feels himself totally unqualified to pass on the correctness of these claims.

The material in the book may be summarized briefly as an argument that the three races of mankind: the Negro, Mongolian, and the so-called "White" are derived from three different branches as are the three great groups of higher apes—the Orang-Utan, the Gorilla and the Chimpanzee. Further, that the Orang-Utan and the Mongolian have come from a common ancestor as have the Gorilla and Negro, and the Chimpanzee and the so-called "White." The appearance of Mongoloid persons in the population is to be explained by Mongolian ancestors who at some earlier time in the history of the race have interbred. These Mongoloid individuals are not necessarily of low intelligence. They are, however, felt to have certain mental characteristics in common. The Mongolian idiot represents an individual of the Mongoloid type who is of defective intelligence. There is no clear line of distinction between the Mongolian idiot and the Mongoloid and one finds every gradation of intelligence in this group.

A great many other ideas have developed, particularly of an anthropological nature. It is impossible to discuss these in any detail.

One may pass certain judgments on the book. It is written in a clear, entertaining style, with considerable emotional bias. It is obviously the author's individual views, given to the reader as such. Those who disagree with him are dealt with in somewhat summary fashion. Considerable of the material is highly speculative, and when one examines certain parts, they do not appear to make a very convincing case. To mention a few points which the reviewer would criticize: We are given a statement concerning "A little imbecile Mongoloid girl who habitually went on all fours like a baboon." One immediately thinks of Hjerdlicka's book, "Children who go on All Fours" in which it is stated that such children are, in general, of superior intelligence. The author states that he has never heard or read of a case of dementia praecox arising among pure bred Negroes. It is, of course, never possible to prove that a Negro is of pure blood, and that there may not have been at some time in the past an admixture from some other race, but certainly there are plenty of cases in this country of dementia praecox among Negroes who appear to be of pure Negro blood. Again, the author says, "My impression is that dementia praecox or puberal insanity, allied with definite Chimpanzoid physique, is more frequent among Jews than among so-called (Nordic) Gentiles." Such a statement of impression makes interesting reading, but hardly qualifies as proof. Another quotation is as follows: "Again, the external sexual organs of the microcephalic and 'Ethiopian' idiots, and of cases of dementia praecox, are over- rather than underdeveloped so far as form is concerned." Such a statement does not conform with most of the writings on dementia praecox.

One might give numerous illustrations of this type, but those given above are sufficient.

In the reviewer's opinion the book is to be regarded as an extremely stimulating but highly speculative book, written by an author who is obviously widely informed on his subject, and who sets forth a number of exceedingly interesting hypotheses. There is much of value in the book and it is very stimulating.

Epidemiology — Historical and Experimental. The Herter Lectures for 1931. By Major Greenwood, F.R.S. The University of London. Baltimore: The Johns Hopkins Press. London: Humphrey Milford, Oxford University Press. Price $1.50.

The fact that a man has been selected to deliver lectures, as above designated, at the Johns Hopkins School of Public Health, means that he has achieved an international reputation, and gives assurance that what the lecturer has to say is well worthy of careful consideration.

With some slight changes Dr. Greenwood's lectures are now transcribed and published in a book of eighty pages.

Dr. Greenwood's approach to his subject may be indicated by the following quotations from the introductory portion of the first of his three lectures:

"Epidemiology, the mass aspects of disease, where not the sick individual but the group, the herd, is the unit of observation, is a very old subject of inquiry; it has interested most of the great physicians from the dawn of knowledge to now. He who neglects the teaching of their experience, who despises even the errors of the past is in truth deceiving and deceived; not less than he who, sneering at the technique of his contemporaries and affecting the rôle of scholarly philosophy, supposes that the writings of a past age, the work of men whose intellectual background is unfamiliar to him, whose very language he has never mastered, render modern laboratory investigations superfluous. . . . Scientific epidemiology, the observation of the phenomena of mass disease by men who believed that these were ordered phenomena, not capricious workings of some arbitrary supernatural power, began, like almost everything which makes life worth living, in Ancient Greece."

The lecturer alludes to and compares the epidemiological theories of Hippocrates, Galen, Frascatorious, Sydenham, Gaunt, Henle, Farr, Brownlee, Ross and others.

He describes experimental studies in epidemiology in laboratory colonies of mice and discusses the conformity of such observations with certain mathematical presumptions.

In his third lecture he endeavors to show how such animal experiments and recent conceptions of the application of mathematics to biometry are in accord with actual human epidemic experience.

The book offers an unusual view of the ills to which "the flesh is heir". Of the erudition of the author there can be no question, but he befogs facts which should not be difficult to grasp by injecting in his discourse allusions to matters that are not essential to an understanding of his subject.

He makes the book tiresome to read because he frequently violates an elementary principle of clear diction in presenting different ideas in the same sentence.

The reading of the book left with the reviewer a question as to the extent to which he had digested the author's thoughts. It also served to recall to the reviewer an incident in his personal experience.

At one time he was employed in a scientific bureau of the Government and associated with the late Robert Simpson Woodward in devising new measuring methods for geodetic purposes. The work made it necessary to test the reliability of observations by applying to them the theory of probabilities. Mr. Woodward was then a mathematician with an international reputation and soon afterwards became the head of a department at Columbia University. He subsequently was more widely known as the President of the Carnegie Institution.

Our Government Bureau was famous for mathematicians of various sorts. One of them, a German, had a son who fell ill with typhoid fever. The father's mathematical mind was attracted by the semblance to a curve resulting from the plotting of the readings of the clinical thermometer on the patient's temperature chart. The father undertook "to work out the equation of the curve of typhoid fever", as he called it, imagining that it would be useful in clinical medicine for diagnostic purposes. Mr. Woodward was shown daily the progress in formulating the equation which was constantly becoming more complicated. One day the father appeared very much disconcerted. The boy had had an intestinal hemorrhage and his temperature suddenly dropped to subnormal. As the boy's temperature was up again the next day the father decided to ignore this "accident" in his computations, but soon afterwards ceased to visit us. Some time later on being asked to let us see his equation he replied "I have given that up. Typhoid fever is too capricious."

It is not improbable that one who has had a different sort of a practical experience as a health officer might have a different reaction to these lectures than the reviewer has had. There will be other readers, however, besides the reviewer who will be left wondering if our modern biometrists fully realize, as this old German came to do, the extent to which accidents in a mathematical sense figure in the manifestations of disease and in statistics relating thereto. One certainly does see attempts to apply mathematical tools to such material without due appreciation of the limitations of their practical usefulness for the purpose in view.

Handbook of the Vaccine Treatment of Chronic Rheumatic Diseases. By H. Warren Crowe. New York: Oxford University Press, 1932. Second Edition. Pp. 71. Price 80c.

This is a manual of vaccine therapy for chronic arthritis. Those familiar with the author's first edition of this manual will find in this edition considerable revision of ideas respecting dosage. This affords an important refinement of the therapy leading to better practical results.

A minimum account of the author's theoretical basis for his method is given; which is just as well, for his theories are open to question. But, the clinical results which are given in abundance and detail, with statistical summaries of the results obtained in two arthritic clinics in the hands of other workers, demand serious attention to the method; for no single method of treatment of arthritis known to the reviewer is capable of giving such uniformly successful results.

This is an important contribution to the field of therapy of chronic arthritis, not only for the apparent effectiveness of the method, but also for its simplicity. The method is given in such detail and sufficient practical illustrations of estimating dosage are included that it is unnecessary for the general practitioner who studies this manual carefully to go far astray.